



STIC Search Report

Biotech-Chem Library

STIC Database Tracking Number: 172748

TO: Ralph J Gitomer
Location: REM-3C18
Art Unit: 1655
Friday, December 16, 2005
Case Serial Number: 10/798986

From: Barb O'Bryen
Location: Biotech-Chem Library
Remsen 1a69
Phone: 571-272-2518

BOB
barbara.obryen@uspto.gov

Search Notes

=> fil capl; d que 15; d que 13; fil medl; d que 195; fil embase; d que 1115; d que 1116

FILE 'CAPLUS' ENTERED AT 16:04:57 ON 16 DEC 2005

USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.

PLEASE SEE "HELP USAGETERMS" FOR DETAILS.

COPYRIGHT (C) 2005 AMERICAN CHEMICAL SOCIETY (ACS)

Copyright of the articles to which records in this database refer is held by the publishers listed in the PUBLISHER (PB) field (available for records published or updated in Chemical Abstracts after December 26, 1996), unless otherwise indicated in the original publications. The CA Lexicon is the copyrighted intellectual property of the American Chemical Society and is provided to assist you in searching databases on STN. Any dissemination, distribution, copying, or storing of this information, without the prior written consent of CAS, is strictly prohibited.

FILE COVERS 1907 - 16 Dec 2005 VOL 143 ISS 26
FILE LAST UPDATED: 15 Dec 2005 (20051215/ED)

Effective October 17, 2005, revised CAS Information Use Policies apply. They are available for your review at:

<http://www.cas.org/infopolicy.html>

'OBI' IS DEFAULT SEARCH FIELD FOR 'CAPLUS' FILE

L1	493	SEA	FILE=CAPLUS	ABB=ON	CHIN W?/AU
L2	2192	SEA	FILE=CAPLUS	ABB=ON	KWON S?/AU
L4	16036	SEA	FILE=CAPLUS	ABB=ON	BIOSENSORS/CT
L5	2	SEA	FILE=CAPLUS	ABB=ON	(L1 OR L2) AND L4

inventor
search

L1	493	SEA	FILE=CAPLUS	ABB=ON	CHIN W?/AU
L2	2192	SEA	FILE=CAPLUS	ABB=ON	KWON S?/AU
L3	0	SEA	FILE=CAPLUS	ABB=ON	L1 AND L2

FILE 'MEDLINE' ENTERED AT 16:04:57 ON 16 DEC 2005

FILE LAST UPDATED: 15 DEC 2005 (20051215/UP). FILE COVERS 1950 TO DATE.

On December 11, 2005, the 2006 MeSH terms were loaded.

The MEDLINE reload for 2006 will soon be available. For details on the 2005 reload, enter HELP RLOAD at an arrow prompt (=>).
See also:

<http://www.nlm.nih.gov/mesh/>
http://www.nlm.nih.gov/pubs/techbull/nd04/nd04_mesh.html
http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_med_data_changes.html
http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_2006_MeSH.html

OLDMEDLINE is covered back to 1950.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2006 vocabulary.

Searched by Barb O'Bryen, STIC 2-2518

This file contains CAS Registry Numbers for easy and accurate

```
L77      447 SEA FILE=MEDLINE ABB=ON CHIN W?/AU
L78      540 SEA FILE=MEDLINE ABB=ON KWON S?/AU
L80      3896 SEA FILE=MEDLINE ABB=ON BIOLOGICAL WARFARE+NT/CT
L81      716 SEA FILE=MEDLINE ABB=ON CHEMICAL WARFARE+NT/CT
L82     102328 SEA FILE=MEDLINE ABB=ON ENVIRONMENTAL EXPOSURE+NT/CT
L83     41512 SEA FILE=MEDLINE ABB=ON AIR POLLUTION+NT/CT
L84     176076 SEA FILE=MEDLINE ABB=ON EPITHELIAL CELLS+NT/CT
L85     174367 SEA FILE=MEDLINE ABB=ON EPITHELIUM+NT/CT
L86     269379 SEA FILE=MEDLINE ABB=ON RESPIRATORY SYSTEM+NT/CT
L87      7060 SEA FILE=MEDLINE ABB=ON CILIA/CT
L88      485 SEA FILE=MEDLINE ABB=ON GOBLET CELLS+NT/CT
L89     10594 SEA FILE=MEDLINE ABB=ON MUCINS+NT/CT
L90      8627 SEA FILE=MEDLINE ABB=ON BIOSENSING TECHNIQUES+NT/CT
L93     758406 SEA FILE=MEDLINE ABB=ON CELLS, CULTURED+NT/CT
L95      1 SEA FILE=MEDLINE ABB=ON (L77 OR L78) AND (L80 OR L81 OR L82
OR L83) AND ((L84 OR L85 OR L86 OR L87 OR L88 OR L89 OR L90)
OR L93)
```

FILE 'EMBASE' ENTERED AT 16:04:57 ON 16 DEC 2005
Copyright (c) 2005 Elsevier B.V. All rights reserved.

FILE COVERS 1974 TO 15 Dec 2005 (20051215/ED)

EMBASE has been reloaded. Enter HELP RLOAD for details.

This file contains CAS Registry Numbers for easy and accurate
substance identification.

```
L102     395 SEA FILE=EMBASE ABB=ON CHIN W?/AU
L103     476 SEA FILE=EMBASE ABB=ON KWON S?/AU
L115      0 SEA FILE=EMBASE ABB=ON L102 AND L103
```

```
L102     395 SEA FILE=EMBASE ABB=ON CHIN W?/AU
L103     476 SEA FILE=EMBASE ABB=ON KWON S?/AU
L104     3037 SEA FILE=EMBASE ABB=ON BIOLOGICAL WARFARE/CT
L105     996 SEA FILE=EMBASE ABB=ON CHEMICAL WARFARE/CT
L106     22836 SEA FILE=EMBASE ABB=ON ENVIRONMENTAL EXPOSURE/CT
L107     84207 SEA FILE=EMBASE ABB=ON "AIR AND AIR RELATED PHENOMENA"+NT/CT
L108     3900 SEA FILE=EMBASE ABB=ON RESPIRATORY EPITHELIUM/CT
L109     187739 SEA FILE=EMBASE ABB=ON CELL CULTURE/CT
L110     218 SEA FILE=EMBASE ABB=ON EUKARYOTIC FLAGELLUM/CT
L111     2385 SEA FILE=EMBASE ABB=ON GOBLET CELL/CT
L112     6530 SEA FILE=EMBASE ABB=ON MUCIN/CT
L113     7849 SEA FILE=EMBASE ABB=ON BIOSENSOR+NT/CT
L114     15116 SEA FILE=EMBASE ABB=ON BIOASSAY/CT
L116      0 SEA FILE=EMBASE ABB=ON (L102 OR L103) AND (L104 OR L105 OR
L106 OR L107) AND (L108 OR L109 OR L110 OR L111 OR L112 OR
L113 OR L114)
```

=> fil jic pascal caba biotechno esbio ntis nioshtic enviroeng healsafe inspec
biosis confsci lifesci polluab toxcenter ceaba wpix scisearch

Searched by Barb O'Bryen, STIC 2-2518

FILE 'JICST-EPLUS' ENTERED AT 16:05:30 ON 16 DEC 2005
COPYRIGHT (C) 2005 Japan Science and Technology Agency (JST)

FILE 'PASCAL' ENTERED AT 16:05:30 ON 16 DEC 2005
Any reproduction or dissemination in part or in full,
by means of any process and on any support whatsoever
is prohibited without the prior written agreement of INIST-CNRS.
COPYRIGHT (C) 2005 INIST-CNRS. All rights reserved.

FILE 'CABA' ENTERED AT 16:05:30 ON 16 DEC 2005
COPYRIGHT (C) 2005 CAB INTERNATIONAL (CABI)

FILE 'BIOTECHNO' ENTERED AT 16:05:30 ON 16 DEC 2005
COPYRIGHT (C) 2005 Elsevier Science B.V., Amsterdam. All rights reserved.

FILE 'ESBIOBASE' ENTERED AT 16:05:30 ON 16 DEC 2005
COPYRIGHT (C) 2005 Elsevier Science B.V., Amsterdam. All rights reserved.

FILE 'NTIS' ENTERED AT 16:05:30 ON 16 DEC 2005
Compiled and distributed by the NTIS, U.S. Department of Commerce.
It contains copyrighted material.
All rights reserved. (2005)

FILE 'NIOSHTIC' ENTERED AT 16:05:30 ON 16 DEC 2005
COPYRIGHT (C) 2005 U.S. Secretary of Commerce on Behalf of the U.S. Government

FILE 'ENVIROENG' ENTERED AT 16:05:30 ON 16 DEC 2005
COPYRIGHT (C) 2005 Cambridge Scientific Abstracts (CSA)

FILE 'HEALSAFE' ENTERED AT 16:05:30 ON 16 DEC 2005
COPYRIGHT (C) 2005 Cambridge Scientific Abstracts (CSA)

FILE 'INSPEC' ENTERED AT 16:05:30 ON 16 DEC 2005
Compiled and produced by the IEE in association with FIZ KARLSRUHE
COPYRIGHT 2005 (c) INSTITUTION OF ELECTRICAL ENGINEERS (IEE)

FILE 'BIOSIS' ENTERED AT 16:05:30 ON 16 DEC 2005
Copyright (c) 2005 The Thomson Corporation

FILE 'CONFSCI' ENTERED AT 16:05:30 ON 16 DEC 2005
COPYRIGHT (C) 2005 Cambridge Scientific Abstracts (CSA)

FILE 'LIFESCI' ENTERED AT 16:05:30 ON 16 DEC 2005
COPYRIGHT (C) 2005 Cambridge Scientific Abstracts (CSA)

FILE 'POLLUAB' ENTERED AT 16:05:30 ON 16 DEC 2005
COPYRIGHT (C) 2005 Cambridge Scientific Abstracts (CSA)

FILE 'TOXCENTER' ENTERED AT 16:05:30 ON 16 DEC 2005
COPYRIGHT (C) 2005 ACS

FILE 'CEABA-VTB' ENTERED AT 16:05:30 ON 16 DEC 2005
COPYRIGHT (c) 2005 DECHEMA eV

FILE 'WPIX' ENTERED AT 16:05:30 ON 16 DEC 2005
COPYRIGHT (C) 2005 THE THOMSON CORPORATION

FILE 'SCISEARCH' ENTERED AT 16:05:30 ON 16 DEC 2005
Copyright (c) 2005 The Thomson Corporation

=> d que 162; d que 163

L1 493 SEA FILE=CAPLUS ABB=ON CHIN W?/AU
L2 2192 SEA FILE=CAPLUS ABB=ON KWON S?/AU
L50 3121 SEA L1
L51 7974 SEA L2
L62 2 SEA L50 AND L51

L1 493 SEA FILE=CAPLUS ABB=ON CHIN W?/AU
L2 2192 SEA FILE=CAPLUS ABB=ON KWON S?/AU
L50 3121 SEA L1
L51 7974 SEA L2
L52 2197473 SEA BIOSENSOR# OR SENSOR#
L53 290424 SEA BIOASSAY?
L54 29232 SEA EPITHELI?(3A) (RESPIRATORY OR CILIAT? OR GOBLET?)
L56 955407 SEA (ENVIRONMENT? OR AIR) (2A) (POLLUT? OR MONITOR? OR QUALITY)
L57 158030 SEA AIRBORNE OR AIR BORNE
L58 17285 SEA (BIOLOGICAL OR CHEMICAL) (2A) WARFARE
L63 0 SEA (L50 OR L51) AND (L52 OR L53) AND (L54 OR (L56 OR L57 OR L58))

=> dup rem 195,15,162

FILE 'MEDLINE' ENTERED AT 16:05:42 ON 16 DEC 2005

FILE 'CAPLUS' ENTERED AT 16:05:42 ON 16 DEC 2005
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2005 AMERICAN CHEMICAL SOCIETY (ACS)

FILE 'BIOSIS' ENTERED AT 16:05:42 ON 16 DEC 2005
Copyright (c) 2005 The Thomson Corporation

FILE 'SCISEARCH' ENTERED AT 16:05:42 ON 16 DEC 2005
Copyright (c) 2005 The Thomson Corporation
PROCESSING COMPLETED FOR L95
PROCESSING COMPLETED FOR L5
PROCESSING COMPLETED FOR L62
L127 5 DUP REM L95 L5 L62 (0 DUPLICATES REMOVED)
ANSWER '1' FROM FILE MEDLINE
ANSWERS '2-3' FROM FILE CAPLUS
ANSWER '4' FROM FILE BIOSIS
ANSWER '5' FROM FILE SCISEARCH

=> d iall 1; d ibib ed abs hitind 2-3; d iall 4-5.

L127 ANSWER 1 OF 5 MEDLINE on STN
ACCESSION NUMBER: 2004189161 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14986108
TITLE: Molecular cloning of a cytosolic ascorbate peroxidase cDNA
from cell cultures of sweet potato and its expression in
response to stress.
AUTHOR: Park S-Y; Ryu S-H; Jang I-C; Kwon S-Y; Kim J-G;
Kwak S-S
CORPORATE SOURCE: Laboratory of Environmental Biotechnology, Korea Research
Institute of Bioscience and Biotechnology, Oun-dong 52,

Searched by Barb O'Bryen, STIC 2-2518

SOURCE: Yusong-gu, 305-806 Daejeon, Korea.
Molecular genetics and genomics : MGG, (2004 Apr) 271 (3)
339-46. Electronic Publication: 2004-02-17.
Journal code: 101093320. ISSN: 1617-4615.
PUB. COUNTRY: Germany; Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AY206407
ENTRY MONTH: 200405
ENTRY DATE: Entered STN: 20040416
Last Updated on STN: 20040529
Entered Medline: 20040528

ABSTRACT:

A cDNA encoding a cytosolic ascorbate peroxidase (APX), swAPX1, was isolated from cell cultures of sweet potato (*Ipomoea batatas*) by cDNA library screening, and its expression in the context of various environmental stresses was investigated. swAPX1 contains an ORF of 250 amino acids (27.5 kDa) encoding a protein with a pI value of 5.32. The swAPX1 ORF does not code for a transit peptide, suggesting that the product is a cytosolic isoform. RNA blot analysis showed that swAPX1 gene is expressed in cultured cells and mature leaves, but not in stems, non-storage or storage roots of sweet potato. The level of swAPX1 RNA progressively increased during cell growth in suspension cultures. In leaf tissues, the gene responded differentially to various abiotic stresses, as revealed by RT-PCR analysis. swAPX1 was highly induced in leaves by wounding, and treatment with methyl viologen (50 microM), hydrogen peroxide (440 mM), abscisic acid (ABA; 100 microM) or exposure to high temperature (37 degrees C). In addition, the gene was strongly induced in the leaves following inoculation with a bacterial pathogen (*Pectobacterium chrysanthemi*). These results indicate that swAPX1 may be involved in hydrogen peroxide detoxification and thus help to overcome the oxidative stress induced by abiotic and biotic stresses.

CONTROLLED TERM: Absciscic Acid: PD, pharmacology
Amino Acid Sequence
Blotting, Northern
Cell Division
Cells, Cultured
Cloning, Molecular
Cytosol
*DNA, Complementary: GE, genetics
Environmental Exposure
*Gene Expression Regulation, Enzymologic
Gene Expression Regulation, Plant
Gene Library
Herbicides: PD, pharmacology
Hydrogen Peroxide: PD, pharmacology
*Ipomoea batatas: EN, enzymology
Ipomoea batatas: GE, genetics
Molecular Sequence Data
Oxidants: PD, pharmacology
*Oxidative Stress
Paraquat: PD, pharmacology
*Peroxidases: GE, genetics
Peroxidases: ME, metabolism
Plant Growth Regulators: PD, pharmacology
Plant Leaves: CY, cytology
*Plant Leaves: ME, metabolism
RNA, Plant: GE, genetics
RNA, Plant: ME, metabolism
Research Support, Non-U.S. Gov't

Searched by Barb O'Bryen, STIC 2-2518

Reverse Transcriptase Polymerase Chain Reaction
Sequence Homology, Amino Acid
Temperature
Wounds and Injuries
CAS REGISTRY NO.: 21293-29-8 (Abscisic Acid); 4685-14-7 (Paraquat); 7722-84-1
(Hydrogen Peroxide)
CHEMICAL NAME: 0 (DNA, Complementary); 0 (Herbicides); 0 (Oxidants); 0
(Plant Growth Regulators); 0 (RNA, Plant); EC 1.11.1.
(Peroxidases); EC 1.11.1.11 (ascorbate peroxidase)

L127 ANSWER 2 OF 5 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2004:936410 CAPLUS
DOCUMENT NUMBER: 142:351522
TITLE: Electrical detection of kinase assay using multi
walled-carbon nanotube (MWCNT) nanoelectrode
AUTHOR(S): Lee, Jae Shin; Kim, Do Hyun; Lee, Seok Jae; Park, Jong
Pil; Park, Tae Jung; Lee, Sang Yup; Jung, Dae-Hwan;
Jung, Hee-Tae; Kim, Jin Hee; **Kwon, Seong Ku**
CORPORATE SOURCE: Dept. of Chemical and Biomolecular engineering, and
Center for Ultramicrochemical Process Systems, KAIST,
Yuseong-gu, Daejeon, 305-701, S. Korea
SOURCE: Special Publication - Royal Society of Chemistry
(2004), 297(Micro Total Analysis Systems 2004, Volume
2), 124-126
CODEN: SROCDO; ISSN: 0260-6291
PUBLISHER: Royal Society of Chemistry
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 06 Nov 2004
AB We have demonstrated the use of MWCNT as a nanoscale probe to monitor the
activity of enzyme kinase. To immobilize the substrate peptide using
carbodiimide chemical, plasma or strong acid treatments were used to induce
carboxyl groups on the sidewall of MWCNTs. After the substrate peptide
immobilization, increase of conductance from MWCNT devices was observed. When
peptide modified MWCNTs react with enzyme kinase, conductance decreases by
several orders of magnitude, and this conductance change can be explained
by the phosphorylation reaction of enzyme kinase. When the sample was
incubated with phosphatase to dephosphorylate the substrate peptide,
nearly complete recovery of the conductance signal has been observed and we
can confirm that we have monitored the kinase activity.
CC 9-7 (Biochemical Methods)
IT **Biosensors**
Blood plasma
Carboxyl group
Electric conductivity
Immobilization, molecular or cellular
(elec. detection of kinase assay using multi walled-carbon
nanotube(MWCNT) nanoelectrode)
REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L127 ANSWER 3 OF 5 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2001:224372 CAPLUS
DOCUMENT NUMBER: 134:234036
TITLE: Non- or minimally-invasive monitoring methods using
particle delivery methods
INVENTOR(S): **Kwon, Sung-yun**

PATENT ASSIGNEE(S): Powderject Research Limited, UK
 SOURCE: U.S., 10 pp.
 CODEN: USXXAM
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 2
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6207400	B1	20010327	US 1999-390068	19990903
JP 2002524120	T2	20020806	JP 2000-568385	19990903
US 2001029957	A1	20011018	US 2001-803828	20010312
US 6482604	B2	20021119		
US 2002102625	A1	20020801	US 2001-22633	20011217
US 6602678	B2	20030805		
US 2005064528	A1	20050324	US 2004-499061	20041105
PRIORITY APPLN. INFO.:			US 1998-99157P	P 19980904
			US 1999-390068	A1 19990903
			WO 1999-GB2914	W 19990903
			US 2001-803828	A2 20010312
			US 2001-22633	A1 20011217
			WO 2002-US37604	W 20021213

ED Entered STN: 29 Mar 2001

AB Methods for sampling an analyte present in a biol. system are provided. The methods entail use of particle delivery methods to obtain a sample of an analyte of interest from the system. Using a needleless syringe particle delivery device, lactose powder was delivered to the skin of diabetic patients. A LifeScan glucose detection membrane strip was moistened with hydroel and applied to the powder-injected site for 1 min. The color intensity of the membranes were quantified with a Bio-Rad densitometer. The interstitial glucose levels were compared with blood glucose concns.

IC ICM* C12Q001-54

ICS C12Q001-00; C12Q001-26; C12M001-00

INCL 435014000

CC 9-16 (Biochemical Methods)

Section cross-reference(s): 14

IT Absorbents

Bioassay

Biochemical molecules

Biosensors

Blood analysis

Body fluid

Diabetes mellitus

Hydrogels

Mucous membrane

Particles

Permeability

Sampling

Skin

(non- or minimally-invasive monitoring methods using particle delivery methods)

REFERENCE COUNT: 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

ACCESSION NUMBER: 2004:286506 BIOSIS
 DOCUMENT NUMBER: PREV200400285263
 TITLE: Premature Activation Mechanisms of Trypsin.
 AUTHOR(S): Yang, Kai [Reprint Author]; Ding, Yongxue; Kwon, Soonjo; Chin, Wei-Chun
 CORPORATE SOURCE: The Department of Biological Science, Florida State University, Biology Unit One, Tallahassee, FL, 32306, USA
 SOURCE: FASEB Journal, (2004) Vol. 18, No. 4-5, pp. Abst. 459.23.
 http://www.fasebj.org/. e-file.
 Meeting Info.: FASEB Meeting on Experimental Biology: Translating the Genome. Washington, District of Columbia, USA. April 17-21, 2004. FASEB.
 ISSN: 0892-6638 (ISSN print).
 DOCUMENT TYPE: Conference; (Meeting)
 Conference; Abstract; (Meeting Abstract)
 LANGUAGE: English
 ENTRY DATE: Entered STN: 16 Jun 2004
 Last Updated on STN: 16 Jun 2004
 ABSTRACT: Under normal physiological conditions, trypsin remains inactive as trypsinogen inside the pancreas. Upon entering the small intestine, trypsinogen is converted to active trypsin. Acute pancreatitis is thought to begin with the premature activation of trypsinogen in the pancreas. However, the exact initiating mechanisms of this premature activation are still not clear. In this study we isolated the zymogen granules (ZGs) from pancreatic acinar cells. The pH fluctuations ((pH)G), Ca²⁺ changes ((Ca²⁺)G) and trypsin premature activation inside ZGs were simultaneously monitored with fluorescent probes. We also investigated possible pharmacological inhibitors and blockers for this premature activation process. Our results showed that a sustained increase of intracellular Ca²⁺ could trigger K⁺ influx into ZGs through Ca²⁺-activated K⁺ channels. The influx of K⁺ can mobilize bound Ca²⁺ by K⁺/Ca²⁺ ion-exchange to increase (Ca²⁺)G and bound H⁺ by K⁺/H⁺ ion-exchange to decrease (pH)G. Both the increase of (Ca²⁺)G and the decrease of (pH)G can facilitate trypsinogen autoactivation and stabilize trypsin activity. Our investigations also showed that trypsin premature activation can be activated by (Ca²⁺) above 300 nM and blocked by apamin (100 μM) and TEA (20 mM). This research project was supported by FSU Cornerstone Program and ABMRF.
 CONCEPT CODE: General biology - Symposia, transactions and proceedings 00520
 Cytology - Animal 02506
 Enzymes - General and comparative studies: coenzymes 10802
 Digestive system - Physiology and biochemistry 14004
 Digestive system - Pathology 14006
 Endocrine - General 17002
 Endocrine - Pancreas 17008
 INDEX TERMS: Major Concepts
 Digestive System (Ingestion and Assimilation);
 Enzymology (Biochemistry and Molecular Biophysics)
 INDEX TERMS: Parts, Structures, & Systems of Organisms
 pancreas: digestive system, endocrine system; pancreatic acinar cell: endocrine system, zymogen granules; small intestine: digestive system
 INDEX TERMS: Diseases
 acute pancreatitis: digestive system disease
 Pancreatitis (MeSH)
 INDEX TERMS: Chemicals & Biochemicals
 trypsin [EC 3.4.21.4]: premature activation mechanism
 INDEX TERMS: Miscellaneous Descriptors
 potassium fluoride/calcium ion exchange

REGISTRY NUMBER: 9002-07-7 (trypsin)
9002-07-7 (EC 3.4.21.4)

L127 ANSWER 5 OF 5 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation on
STN

ACCESSION NUMBER: 2004:619852 SCISEARCH

THE GENUINE ARTICLE: 806ZA

TITLE: Premature activation mechanisms of trypsin

AUTHOR: Yang K (Reprint); Ding Y X; Kwon S; Chin W
C

CORPORATE SOURCE: Florida State Univ, Dept Biol Sci, Biol Unit 1,
Tallahassee, FL 32306 USA; Florida State Univ, Dept Chem
Engn, Tallahassee, FL 32306 USA; Florida State Univ,
Biomed Engn Program, Tallahassee, FL 32306 USA

COUNTRY OF AUTHOR: USA

SOURCE: FASEB JOURNAL, (23 MAR 2004) Vol. 18, No. 4, Supp. [S],
pp. A705-A705.
ISSN: 0892-6638.

PUBLISHER: FEDERATION AMER SOC EXP BIOL, 9650 ROCKVILLE PIKE,
BETHESDA, MD 20814-3998 USA.

DOCUMENT TYPE: Conference; Journal

LANGUAGE: English

REFERENCE COUNT: 0

ENTRY DATE: Entered STN: 29 Jul 2004
Last Updated on STN: 29 Jul 2004

CATEGORY: BIOCHEMISTRY & MOLECULAR BIOLOGY; BIOLOGY; CELL BIOLOGY

=> fil embase; d que l118; d que l123; d que l126

FILE 'EMBASE' ENTERED AT 16:08:34 ON 16 DEC 2005
Copyright (c) 2005 Elsevier B.V. All rights reserved.

FILE COVERS 1974 TO 15 Dec 2005 (20051215/ED)

EMBASE has been reloaded. Enter HELP RLOAD for details.

This file contains CAS Registry Numbers for easy and accurate
substance identification.

L104	3037	SEA FILE=EMBASE ABB=ON	BIOLOGICAL WARFARE/CT
L105	996	SEA FILE=EMBASE ABB=ON	CHEMICAL WARFARE/CT
L106	22836	SEA FILE=EMBASE ABB=ON	ENVIRONMENTAL EXPOSURE/CT
L107	84207	SEA FILE=EMBASE ABB=ON	"AIR AND AIR RELATED PHENOMENA"+NT/CT
L108	3900	SEA FILE=EMBASE ABB=ON	RESPIRATORY EPITHELIUM/CT
L113	7849	SEA FILE=EMBASE ABB=ON	BIOSENSOR+NT/CT
L114	15116	SEA FILE=EMBASE ABB=ON	BIOASSAY/CT
L118	1	SEA FILE=EMBASE ABB=ON	(L104 OR L105 OR L106 OR L107) AND L108 AND (L113 OR L114)

L104	3037	SEA FILE=EMBASE ABB=ON	BIOLOGICAL WARFARE/CT
L105	996	SEA FILE=EMBASE ABB=ON	CHEMICAL WARFARE/CT
L106	22836	SEA FILE=EMBASE ABB=ON	ENVIRONMENTAL EXPOSURE/CT
L107	84207	SEA FILE=EMBASE ABB=ON	"AIR AND AIR RELATED PHENOMENA"+NT/CT
L108	3900	SEA FILE=EMBASE ABB=ON	RESPIRATORY EPITHELIUM/CT
L109	187739	SEA FILE=EMBASE ABB=ON	CELL CULTURE/CT
L119	578914	SEA FILE=EMBASE ABB=ON	IN VITRO STUDY/CT
L121	4485	SEA FILE=EMBASE ABB=ON	MONOLAYER CULTURE/CT
L122	1551	SEA FILE=EMBASE ABB=ON	CILIARY MOTILITY/CT
L123	4	SEA FILE=EMBASE ABB=ON	(L104 OR L105 OR L106 OR L107) AND L108 AND L109 AND (L119 OR (L121 OR L122))

L104	3037	SEA FILE=EMBASE ABB=ON	BIOLOGICAL WARFARE/CT
L105	996	SEA FILE=EMBASE ABB=ON	CHEMICAL WARFARE/CT
L106	22836	SEA FILE=EMBASE ABB=ON	ENVIRONMENTAL EXPOSURE/CT
L107	84207	SEA FILE=EMBASE ABB=ON	"AIR AND AIR RELATED PHENOMENA"+NT/CT
L108	3900	SEA FILE=EMBASE ABB=ON	RESPIRATORY EPITHELIUM/CT
L109	187739	SEA FILE=EMBASE ABB=ON	CELL CULTURE/CT
L117	12	SEA FILE=EMBASE ABB=ON	(L104 OR L105 OR L106 OR L107) AND L108 AND L109
L125	38308	SEA FILE=EMBASE ABB=ON	QUANTITATIVE ANALYSIS/CT
L126	1	SEA FILE=EMBASE ABB=ON	L117 AND L125

=> s l118 or l123 or l126

L128 6 L118 OR L123 OR L126

=> fil capl; d que l7; d que l27; d que l28; d que l30; d que l37

FILE 'CAPLUS' ENTERED AT 16:08:36 ON 16 DEC 2005
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.

PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2005 AMERICAN CHEMICAL SOCIETY (ACS)

Copyright of the articles to which records in this database refer is held by the publishers listed in the PUBLISHER (PB) field (available for records published or updated in Chemical Abstracts after December 26, 1996), unless otherwise indicated in the original publications. The CA Lexicon is the copyrighted intellectual property of the American Chemical Society and is provided to assist you in searching databases on STN. Any dissemination, distribution, copying, or storing of this information, without the prior written consent of CAS, is strictly prohibited.

FILE COVERS 1907 - 16 Dec 2005 VOL 143 ISS 26
FILE LAST UPDATED: 15 Dec 2005 (20051215/ED)

Effective October 17, 2005, revised CAS Information Use Policies apply. They are available for your review at:

<http://www.cas.org/infopolicy.html>
'OBI' IS DEFAULT SEARCH FIELD FOR 'CAPLUS' FILE

L4 16036 SEA FILE=CAPLUS ABB=ON BIOSENSORS/CT
L6 3734 SEA FILE=CAPLUS ABB=ON EPIITHELI?/OBI (L) (RESPIRATORY/OBI OR
CILIAT?/OBI OR GOBLET?/OBI)
L7 1 SEA FILE=CAPLUS ABB=ON L4 AND L6

L6 3734 SEA FILE=CAPLUS ABB=ON EPIITHELI?/OBI (L) (RESPIRATORY/OBI OR
CILIAT?/OBI OR GOBLET?/OBI)
L8 208880 SEA FILE=CAPLUS ABB=ON CULTUR?/OBI
L9 244 SEA FILE=CAPLUS ABB=ON L6 AND L8
L10 42474 SEA FILE=CAPLUS ABB=ON ENVIRONMENTAL POLLUTION/CT
L11 147315 SEA FILE=CAPLUS ABB=ON AIR/OBI (L) POLLUTION/CW
L12 4705 SEA FILE=CAPLUS ABB=ON (BIOLOGICAL/OBI OR CHEMICAL/OBI) (L) WARF
ARE/OBI
L13 13214 SEA FILE=CAPLUS ABB=ON AIR/OBI (L) MONITOR?/OBI
L26 20819 SEA FILE=CAPLUS ABB=ON SAMPLING/CW
L27 4 SEA FILE=CAPLUS ABB=ON L9 AND ((L10 OR L11 OR L12 OR L13) OR
L26)

L6 3734 SEA FILE=CAPLUS ABB=ON EPIITHELI?/OBI (L) (RESPIRATORY/OBI OR
CILIAT?/OBI OR GOBLET?/OBI)
L10 42474 SEA FILE=CAPLUS ABB=ON ENVIRONMENTAL POLLUTION/CT
L11 147315 SEA FILE=CAPLUS ABB=ON AIR/OBI (L) POLLUTION/CW
L12 4705 SEA FILE=CAPLUS ABB=ON (BIOLOGICAL/OBI OR CHEMICAL/OBI) (L) WARF
ARE/OBI
L13 13214 SEA FILE=CAPLUS ABB=ON AIR/OBI (L) MONITOR?/OBI
L16 11248 SEA FILE=CAPLUS ABB=ON AIRBORNE PARTICLES/CT
L17 56518 SEA FILE=CAPLUS ABB=ON TOXICITY/CT
L18 4102 SEA FILE=CAPLUS ABB=ON ECOTOXICITY/CT
L19 9069 SEA FILE=CAPLUS ABB=ON BIOASSAY/CT
L22 14 SEA FILE=CAPLUS ABB=ON L6 AND L19
L26 20819 SEA FILE=CAPLUS ABB=ON SAMPLING/CW
L28 3 SEA FILE=CAPLUS ABB=ON L22 AND ((L10 OR L11 OR L12 OR L13) OR

L26 OR (L16 OR L17 OR L18))

L4 16036 SEA FILE=CAPLUS ABB=ON BIOSENSORS/CT
 L6 3734 SEA FILE=CAPLUS ABB=ON EPITHELI?/OBI (L) (RESPIRATORY/OBI OR
 CILIAT?/OBI OR GOBLET?/OBI)
 L7 1 SEA FILE=CAPLUS ABB=ON L4 AND L6
 L8 208880 SEA FILE=CAPLUS ABB=ON CULTUR?/OBI
 L9 244 SEA FILE=CAPLUS ABB=ON L6 AND L8
 L10 42474 SEA FILE=CAPLUS ABB=ON ENVIRONMENTAL POLLUTION/CT.
 L11 147315 SEA FILE=CAPLUS ABB=ON AIR/OBI (L) POLLUTION/CW
 L12 4705 SEA FILE=CAPLUS ABB=ON (BIOLOGICAL/OBI OR CHEMICAL/OBI) (L) WARF
 ARE/OBI
 L13 13214 SEA FILE=CAPLUS ABB=ON AIR/OBI (L) MONITOR?/OBI
 L16 11248 SEA FILE=CAPLUS ABB=ON AIRBORNE PARTICLES/CT
 L17 56518 SEA FILE=CAPLUS ABB=ON TOXICITY/CT
 L18 4102 SEA FILE=CAPLUS ABB=ON ECOTOXICITY/CT
 L19 9069 SEA FILE=CAPLUS ABB=ON BIOASSAY/CT
 L20 427058 SEA FILE=CAPLUS ABB=ON 59/SC, SX - *Section code - Air pollution & industrial hygiene*
 L21 615834 SEA FILE=CAPLUS ABB=ON 4/SC, SX - *Section code - Toxicology*
 L22 14 SEA FILE=CAPLUS ABB=ON L6 AND L19
 L25 74 SEA FILE=CAPLUS ABB=ON L8 AND L20 AND L21 AND ((L10 OR L11 OR
 L12 OR L13) OR L16)
 L26 20819 SEA FILE=CAPLUS ABB=ON SAMPLING/CW
 L27 4 SEA FILE=CAPLUS ABB=ON L9 AND ((L10 OR L11 OR L12 OR L13) OR
 L26)
 L28 3 SEA FILE=CAPLUS ABB=ON L22 AND ((L10 OR L11 OR L12 OR L13) OR
 L26 OR (L16 OR L17 OR L18))
 L29 3 SEA FILE=CAPLUS ABB=ON L25 AND L26
 L30 3 SEA FILE=CAPLUS ABB=ON L29 NOT (L7 OR L27 OR L28)

L4 16036 SEA FILE=CAPLUS ABB=ON BIOSENSORS/CT
 L6 3734 SEA FILE=CAPLUS ABB=ON EPITHELI?/OBI (L) (RESPIRATORY/OBI OR
 CILIAT?/OBI OR GOBLET?/OBI)
 L10 42474 SEA FILE=CAPLUS ABB=ON ENVIRONMENTAL POLLUTION/CT
 L11 147315 SEA FILE=CAPLUS ABB=ON AIR/OBI (L) POLLUTION/CW
 L12 4705 SEA FILE=CAPLUS ABB=ON (BIOLOGICAL/OBI OR CHEMICAL/OBI) (L) WARF
 ARE/OBI
 L13 13214 SEA FILE=CAPLUS ABB=ON AIR/OBI (L) MONITOR?/OBI
 L16 11248 SEA FILE=CAPLUS ABB=ON AIRBORNE PARTICLES/CT
 L17 56518 SEA FILE=CAPLUS ABB=ON TOXICITY/CT
 L18 4102 SEA FILE=CAPLUS ABB=ON ECOTOXICITY/CT
 L19 9069 SEA FILE=CAPLUS ABB=ON BIOASSAY/CT
 L20 427058 SEA FILE=CAPLUS ABB=ON 59/SC, SX
 L21 615834 SEA FILE=CAPLUS ABB=ON 4/SC, SX
 L26 20819 SEA FILE=CAPLUS ABB=ON SAMPLING/CW
 L31 647 SEA FILE=CAPLUS ABB=ON (CILIA? (2A) BEAT?)/BI
 L32 2600 SEA FILE=CAPLUS ABB=ON (ELECTRICAL RESPON?)/BI
 L33 1144 SEA FILE=CAPLUS ABB=ON (SECRET? (2A) MUCIN#)/BI
 L37 8 SEA FILE=CAPLUS ABB=ON L6 AND (L31 OR L32 OR L33) AND (L4 OR
 (L10 OR L11 OR L12 OR L13) OR (L16 OR L17 OR L18 OR L19 OR
 L20) OR L26) AND L21

=> s (l7 or l27 or l28 or l30 or l37) not l5

L129 18 (L7 OR L27 OR L28 OR L30 OR L37) NOT (L5) *previously printed w/ inventor search*

=> fil medl; d que l101; s l101 not l95

FILE 'MEDLINE' ENTERED AT 16:08:38 ON 16 DEC 2005

FILE LAST UPDATED: 15 DEC 2005 (20051215/UP). FILE COVERS 1950 TO DATE.

On December 11, 2005, the 2006 MeSH terms were loaded.

The MEDLINE reload for 2006 will soon be available. For details on the 2005 reload, enter HELP RLOAD at an arrow prompt (=>).
See also:

<http://www.nlm.nih.gov/mesh/>
http://www.nlm.nih.gov/pubs/techbull/nd04/nd04_mesh.html
http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_med_data_changes.html
http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_2006_MeSH.html

OLDMEDLINE is covered back to 1950.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2006 vocabulary.

This file contains CAS Registry Numbers for easy and accurate

L80	3896	SEA	FILE=MEDLINE	ABB=ON	BIOLOGICAL WARFARE+NT/CT
L81	716	SEA	FILE=MEDLINE	ABB=ON	CHEMICAL WARFARE+NT/CT
L82	102328	SEA	FILE=MEDLINE	ABB=ON	ENVIRONMENTAL EXPOSURE+NT/CT
L83	41512	SEA	FILE=MEDLINE	ABB=ON	AIR POLLUTION+NT/CT
L84	176076	SEA	FILE=MEDLINE	ABB=ON	EPITHELIAL CELLS+NT/CT
L85	174367	SEA	FILE=MEDLINE	ABB=ON	EPITHELIUM+NT/CT
L86	269379	SEA	FILE=MEDLINE	ABB=ON	RESPIRATORY SYSTEM+NT/CT
L87	7060	SEA	FILE=MEDLINE	ABB=ON	CILIA/CT
L88	485	SEA	FILE=MEDLINE	ABB=ON	GOBLET CELLS+NT/CT
L89	10594	SEA	FILE=MEDLINE	ABB=ON	MUCINS+NT/CT
L93	758406	SEA	FILE=MEDLINE	ABB=ON	CELLS, CULTURED+NT/CT
L100	60664	SEA	FILE=MEDLINE	ABB=ON	AIR POLLUTANTS+NT/CT
L101	9	SEA	FILE=MEDLINE	ABB=ON	((L80 OR L81 OR L82 OR L83) OR L100)
					AND (L84 OR L85) AND L86 AND L93 AND (L87 OR L88 OR L89)

L130

9 L101 NOT (L95)

previously printed

=> fil jic pascal caba biotechno esbio ntis nioshtic enviroeng healsafe inspec
biosis confsci lifesci polluab toxcenter ceaba wpix scisearch
FILE 'JICST-EPLUS' ENTERED AT 16:08:50 ON 16 DEC 2005
COPYRIGHT (C) 2005 Japan Science and Technology Agency (JST)

FILE 'PASCAL' ENTERED AT 16:08:50 ON 16 DEC 2005
Any reproduction or dissemination in part or in full,
by means of any process and on any support whatsoever
is prohibited without the prior written agreement of INIST-CNRS.
COPYRIGHT (C) 2005 INIST-CNRS. All rights reserved.

FILE 'CABA' ENTERED AT 16:08:50 ON 16 DEC 2005
COPYRIGHT (C) 2005 CAB INTERNATIONAL (CABI)

FILE 'BIOTECHNO' ENTERED AT 16:08:50 ON 16 DEC 2005

COPYRIGHT (C) 2005 Elsevier Science B.V., Amsterdam. All rights reserved.

FILE 'ESBIOBASE' ENTERED AT 16:08:50 ON 16 DEC 2005
COPYRIGHT (C) 2005 Elsevier Science B.V., Amsterdam. All rights reserved.

FILE 'NTIS' ENTERED AT 16:08:50 ON 16 DEC 2005
Compiled and distributed by the NTIS, U.S. Department of Commerce.
It contains copyrighted material.
All rights reserved. (2005)

FILE 'NIOSHTIC' ENTERED AT 16:08:50 ON 16 DEC 2005
COPYRIGHT (C) 2005 U.S. Secretary of Commerce on Behalf of the U.S. Government

FILE 'ENVIROENG' ENTERED AT 16:08:50 ON 16 DEC 2005
COPYRIGHT (C) 2005 Cambridge Scientific Abstracts (CSA)

FILE 'HEALSAFE' ENTERED AT 16:08:50 ON 16 DEC 2005
COPYRIGHT (C) 2005 Cambridge Scientific Abstracts (CSA)

FILE 'INSPEC' ENTERED AT 16:08:50 ON 16 DEC 2005
Compiled and produced by the IEE in association with FIZ KARLSRUHE
COPYRIGHT 2005 (c) INSTITUTION OF ELECTRICAL ENGINEERS (IEE)

FILE 'BIOSIS' ENTERED AT 16:08:50 ON 16 DEC 2005
Copyright (c) 2005 The Thomson Corporation

FILE 'CONFSCI' ENTERED AT 16:08:50 ON 16 DEC 2005
COPYRIGHT (C) 2005 Cambridge Scientific Abstracts (CSA)

FILE 'LIFESCI' ENTERED AT 16:08:50 ON 16 DEC 2005
COPYRIGHT (C) 2005 Cambridge Scientific Abstracts (CSA)

FILE 'POLLUAB' ENTERED AT 16:08:50 ON 16 DEC 2005
COPYRIGHT (C) 2005 Cambridge Scientific Abstracts (CSA)

FILE 'TOXCENTER' ENTERED AT 16:08:50 ON 16 DEC 2005
COPYRIGHT (C) 2005 ACS

FILE 'CEABA-VTB' ENTERED AT 16:08:50 ON 16 DEC 2005
COPYRIGHT (c) 2005 DECHEMA eV

FILE 'WPIX' ENTERED AT 16:08:50 ON 16 DEC 2005
COPYRIGHT (C) 2005 THE THOMSON CORPORATION

FILE 'SCISEARCH' ENTERED AT 16:08:50 ON 16 DEC 2005
Copyright (c) 2005 The Thomson Corporation

=> d que 166; d que 167; d que 168; d que 173; d que 174

L52 2197473 SEA BIOSENSOR# OR SENSOR#
L53 290424 SEA BIOASSAY?
L54 29232 SEA EPITHELI?(3A) (RESPIRATORY OR CILIAT? OR GOBLET?)
L55 3624943 SEA CULTUR?
L56 955407 SEA (ENVIRONMENT? OR AIR) (2A) (POLLUT? OR MONITOR? OR QUALITY)
L57 158030 SEA AIRBORNE OR AIR BORNE
L58 17285 SEA (BIOLOGICAL OR CHEMICAL) (2A) WARFARE
L65 122 SEA (L52 OR L53) AND L54 AND L55
L66 16 SEA L65 AND (L56 OR L57 OR L58)

L54 29232 SEA EPITHELI? (3A) (RESPIRATORY OR CILIAT? OR GOBLET?)
 L55 3624943 SEA CULTUR?
 L56 955407 SEA (ENVIRONMENT? OR AIR) (2A) (POLLUT? OR MONITOR? OR QUALITY)
 L57 158030 SEA AIRBORNE OR AIR BORNE
 L58 17285 SEA (BIOLOGICAL OR CHEMICAL) (2A) WARFARE
 L59 4285 SEA (CILIA? (2A) BEAT?)
 L60 7049 SEA (ELECTRICAL RESPON?)
 L61 4997 SEA (SECRET? (2A) MUCIN#)
 L67 2 SEA L54 (5A) L55 AND (L56 OR L57 OR L58) AND (L59 OR L60 OR L61)

L52 2197473 SEA BIOSENSOR# OR SENSOR#
 L53 290424 SEA BIOASSAY?
 L54 29232 SEA EPITHELI? (3A) (RESPIRATORY OR CILIAT? OR GOBLET?)
 L56 955407 SEA (ENVIRONMENT? OR AIR) (2A) (POLLUT? OR MONITOR? OR QUALITY)
 L57 158030 SEA AIRBORNE OR AIR BORNE
 L58 17285 SEA (BIOLOGICAL OR CHEMICAL) (2A) WARFARE
 L59 4285 SEA (CILIA? (2A) BEAT?)
 L60 7049 SEA (ELECTRICAL RESPON?)
 L61 4997 SEA (SECRET? (2A) MUCIN#)
 L64 1378 SEA (L52 OR L53) AND L54
 L68 1 SEA L64 AND (L56 OR L57 OR L58) AND (L59 OR L60 OR L61)

L52 2197473 SEA BIOSENSOR# OR SENSOR#
 L53 290424 SEA BIOASSAY?
 L54 29232 SEA EPITHELI? (3A) (RESPIRATORY OR CILIAT? OR GOBLET?)
 L59 4285 SEA (CILIA? (2A) BEAT?)
 L60 7049 SEA (ELECTRICAL RESPON?)
 L61 4997 SEA (SECRET? (2A) MUCIN#)
 L64 1378 SEA (L52 OR L53) AND L54
 L69 32 SEA L64 AND (L59 OR L60 OR L61)
 L71 1500078 SEA TOXICITY/CT OR PHARMACOLOGICAL OR SCREEN?/TI
 L72 4696113 SEA SAMPL?
 L73 9 SEA L69 AND (L71 OR L72)

L54 29232 SEA EPITHELI? (3A) (RESPIRATORY OR CILIAT? OR GOBLET?)
 L55 3624943 SEA CULTUR?
 L56 955407 SEA (ENVIRONMENT? OR AIR) (2A) (POLLUT? OR MONITOR? OR QUALITY)
 L57 158030 SEA AIRBORNE OR AIR BORNE
 L58 17285 SEA (BIOLOGICAL OR CHEMICAL) (2A) WARFARE
 L72 4696113 SEA SAMPL?
 L74 6 SEA L54 (5A) L55 AND L72 AND (L56 OR L57 OR L58)

=> s 166-168
 L131 19 (L66 OR L67 OR L68)

=> s 173-174
 L132 15 (L73 OR L74)

=> s l131-l132
L135 34 (L131 OR L132)

=> dup rem l130,l129,l128,l135
FILE 'MEDLINE' ENTERED AT 16:10:42 ON 16 DEC 2005

FILE 'CAPLUS' ENTERED AT 16:10:42 ON 16 DEC 2005
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2005 AMERICAN CHEMICAL SOCIETY (ACS)

FILE 'EMBASE' ENTERED AT 16:10:42 ON 16 DEC 2005
Copyright (c) 2005 Elsevier B.V. All rights reserved.

FILE 'PASCAL' ENTERED AT 16:10:42 ON 16 DEC 2005
Any reproduction or dissemination in part or in full,
by means of any process and on any support whatsoever
is prohibited without the prior written agreement of INIST-CNRS.
COPYRIGHT (C) 2005 INIST-CNRS. All rights reserved.

FILE 'ESBIOBASE' ENTERED AT 16:10:42 ON 16 DEC 2005
COPYRIGHT (C) 2005 Elsevier Science B.V., Amsterdam. All rights reserved.

FILE 'NIOSHTIC' ENTERED AT 16:10:42 ON 16 DEC 2005
COPYRIGHT (C) 2005 U.S. Secretary of Commerce on Behalf of the U.S. Government

FILE 'INSPEC' ENTERED AT 16:10:42 ON 16 DEC 2005
Compiled and produced by the IEE in association with FIZ KARLSRUHE
COPYRIGHT 2005 (c) INSTITUTION OF ELECTRICAL ENGINEERS (IEE)

FILE 'BIOSIS' ENTERED AT 16:10:42 ON 16 DEC 2005
Copyright (c) 2005 The Thomson Corporation

FILE 'LIFESCI' ENTERED AT 16:10:42 ON 16 DEC 2005
COPYRIGHT (C) 2005 Cambridge Scientific Abstracts (CSA)

FILE 'TOXCENTER' ENTERED AT 16:10:42 ON 16 DEC 2005
COPYRIGHT (C) 2005 ACS

FILE 'SCISEARCH' ENTERED AT 16:10:42 ON 16 DEC 2005
Copyright (c) 2005 The Thomson Corporation
PROCESSING COMPLETED FOR L130
PROCESSING COMPLETED FOR L129
PROCESSING COMPLETED FOR L128
PROCESSING COMPLETED FOR L135
L136 47 DUP REM L130 L129 L128 L135 (20 DUPLICATES REMOVED)
ANSWERS '1-9' FROM FILE MEDLINE
ANSWERS '10-26' FROM FILE CAPLUS
ANSWERS '27-31' FROM FILE EMBASE
ANSWERS '32-33' FROM FILE PASCAL
ANSWERS '34-36' FROM FILE ESBIOBASE
ANSWER '37' FROM FILE NIOSHTIC
ANSWER '38' FROM FILE INSPEC
ANSWERS '39-43' FROM FILE BIOSIS
ANSWERS '44-47' FROM FILE TOXCENTER

=> d iall 1-9; d ibib ed abs hitind 10-26; d iall 27-47; fil hom

Gene Expression: DE, drug effects
 Guinea Pigs
 *Industrial Waste
 Metals: TO, toxicity
 Mucins: BI, biosynthesis
 Mucins: GE, genetics
 *Mucins: SE, secretion
 Oxidants: PD, pharmacology
 RNA, Messenger: BI, biosynthesis
 RNA, Messenger: GE, genetics
 Research Support, U.S. Gov't, Non-P.H.S.
 Research Support, U.S. Gov't, P.H.S.
 Trachea: CY, cytology
 Trachea: DE, drug effects
 *Trachea: ME, metabolism

CAS REGISTRY NO.: 68131-74-8 (fly ash); 7440-44-0 (Carbon)
 CHEMICAL NAME: 0 (Antioxidants); 0 (Industrial Waste); 0 (Metals); 0 (Mucins); 0 (Oxidants); 0 (RNA, Messenger)

L136 ANSWER 2 OF 47 MEDLINE on STN DUPLICATE 10
 ACCESSION NUMBER: 93076221 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 1446252
 TITLE: Tracheal epithelium in culture: a model for toxicity testing of inhaled molecules.
 AUTHOR: Romet-Haddad S; Marano F; Blanquart C; Baeza-Squiban A
 CORPORATE SOURCE: Laboratoire de Cytophysiologie et Toxicologie Cellulaire, Universite Paris, France.
 SOURCE: Cell biology and toxicology, (1992 Jul-Sep) 8 (3) 141-50.
 Journal code: 8506639. ISSN: 0742-2091.
 PUB. COUNTRY: Netherlands
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 199212
 ENTRY DATE: Entered STN: 19930129
 Last Updated on STN: 19930129
 Entered Medline: 19921229

ABSTRACT:

Rabbit trachea primary cultures have been developed as a model to evaluate the toxicity of noxious airborne pollutants. A mucociliary epithelium has been restored in vitro on collagen gel. Several general cytotoxicity assays (viability and growth inhibition) permit a first assessment for the acute toxicity of the tested molecules. More specific criteria such as measurement of the integrity of the epithelial barrier and inhibition of ciliary beat frequency allow to determine a specific impact of xenobiotics on the mucociliary epithelium in culture.

CONTROLLED TERM: Acrolein: TO, toxicity
 *Air Pollutants, Environmental: TO, toxicity
 Animals
 Cell Division: DE, drug effects
 Cell Survival: DE, drug effects
 Cells, Cultured
 Cilia: DE, drug effects
 Electrodes
 Epithelial Cells
 Epithelium: DE, drug effects
 Mechlorethamine: TO, toxicity
 *Models, Biological
 Rabbits
 Research Support, Non-U.S. Gov't

L136 ANSWER 1 OF 47 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 2000169401 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10702361
TITLE: Residual oil fly ash induces cytotoxicity and mucin
secretion by guinea pig tracheal epithelial cells via an
oxidant-mediated mechanism.
AUTHOR: Jiang N; Dreher K L; Dye J A; Li Y; Richards J H; Martin L
D; Adler K B
CORPORATE SOURCE: Department of Anatomy, College of Veterinary Medicine,
North Carolina State University, Raleigh, North Carolina,
USA.
CONTRACT NUMBER: HL 36982 (NHLBI)
SOURCE: Toxicology and applied pharmacology, (2000 Mar 15) 163 (3)
221-30.
Journal code: 0416575. ISSN: 0041-008X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200004
ENTRY DATE: Entered STN: 20000421
Last Updated on STN: 20021210
Entered Medline: 20000410

ABSTRACT:
Inhalation of ambient air particulate matter (PM) is associated with pulmonary injury and inflammation. Using primary cultures of guinea pig tracheal epithelial (GPTE) cells as an in vitro model of airway epithelium, we examined effects of exposure to suspensions of six different emission and ambient air PM samples: residual oil fly ash (ROFA) from an electrical power plant; fly ash from a domestic oil burning furnace (DOFA); ambient air dust from St. Louis (STL), Ottawa (OT), and Washington, DC (WDC); and volcanic ash from the eruption of Mount Saint Helens (MSH) in 1980. Effects of these particulates on cell viability (assessed via LDH assay), secretion of mucin (measured by a monoclonal antibody-based ELISA), and steady-state mRNA levels of the mucin gene MUC2 were determined. ROFA was the most toxic of the dusts tested, as it significantly increased LDH release following a 24-h incubation with 50 microg/cm(2) ROFA. ROFA also enhanced MUC2 mRNA after 4-h exposure, and mucin secretion after 8 h. ROFA-induced mucin secretion and cytotoxicity were attenuated by the oxidant scavenger, dimethylthiourea (DMTU). ROFA exposure also depleted cells of glutathione (GSH). Relatedly, depletion of intracellular GSH by treatment of the cells with buthionine sulfoxamine (BSO) also provoked mucin secretion, as well as enhancing the secretory effect of ROFA when the two agents were added together. L-NMA, the nitric oxide synthase (NOS) inhibitor, did not affect ROFA-induced mucin secretion. Of the soluble transition metals in ROFA (nickel, iron, vanadium), only vanadium individually, or combinations of the metals containing vanadium, provoked secretion. The results suggest ROFA enhances mucin secretion and generates toxicity in vitro to airway epithelium via a mechanism(s) involving generation of oxidant stress, perhaps related to depletion of cellular antioxidant capacity. Deleterious effects of inhalation of ROFA in the respiratory tract in vivo may relate to these cellular responses. Vanadium, a component of ROFA, may be important in generating these reactions.

Copyright 2000 Academic Press.

CONTROLLED TERM: Animals
Antioxidants: PD, pharmacology
*Carbon: TO, toxicity
Cell Survival: DE, drug effects
Cells, Cultured
Dust: AE, adverse effects
Epithelial Cells: DE, drug effects

Searched by Barb O'Bryen, STIC 2-2518

Trachea: CY, cytology
 *Trachea: DE, drug effects
 CAS REGISTRY NO.: 107-02-8 (Acrolein); 51-75-2 (Mechlorethamine)
 CHEMICAL NAME: 0 (Air Pollutants, Environmental)

L136 ANSWER 3 OF 47 MEDLINE on STN
 ACCESSION NUMBER: 2003057681 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 12568494
 TITLE: Mechanisms by which gram-positive bacteria and tobacco smoke stimulate mucin induction through the epidermal growth factor receptor (EGFR).
 AUTHOR: Basbaum Carol; Li Daizong; Gensch Erin; Gallup Marianne; Lemjabbar Hassan
 CORPORATE SOURCE: Biomedical Sciences Program, Department of Anatomy, University of California, San Francisco, CA 94143-0452, USA.
 SOURCE: Novartis Foundation symposium, (2002) 248 171-6; discussion 176-80, 277-82.
 Journal code: 9807767. ISSN: 1528-2511.
 PUB. COUNTRY: England: United Kingdom
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 200305
 ENTRY DATE: Entered STN: 20030206
 Last Updated on STN: 20030529
 Entered Medline: 20030528

ABSTRACT:
 Mucin, the major macromolecular component of mucus, is generally considered to be a protective substance. When overproduced in a variety of lung diseases, however, mucin gives rise to clinical problems such as airway obstruction and recurrent infection. Our approach to identifying drug targets for the control of mucin overproduction is the analysis of cellular signalling pathways linking stimuli in the diseased lung to mucin transcription. Here we show that mucin transcription in response to both gram-positive bacteria and tobacco smoke is mediated through activation of the epidermal growth factor receptor (EGFR). The mode of activation of EGFR in response to bacterial lipoteichoic acid involves cleavage of the transmembrane ligand HBEGF by ADAM 10, whereas the activation of EGFR in response to smoke involves cleavage of amphiregulin by ADAM 17.

CONTROLLED TERM: Cell Line
 Endopeptidases: PH, physiology
 Epithelial Cells: DE, drug effects
 Epithelial Cells: ME, metabolism
 Gene Expression Regulation: PH, physiology
 Glycoproteins: ME, metabolism
 *Gram-Positive Bacteria: PH, physiology
 Humans
 Intercellular Signaling Peptides and Proteins: ME, metabolism
 Ligands
 Lipopolysaccharides: PD, pharmacology
 Lung: DE, drug effects
 *Lung: ME, metabolism
 Metalloendopeptidases: PH, physiology
 *Mucins: BI, biosynthesis
 Mucins: GE, genetics
 Phosphorylation
 Protein Processing, Post-Translational
 Receptor, Epidermal Growth Factor: DE, drug effects

Searched by Barb O'Bryen, STIC 2-2518

*Receptor, Epidermal Growth Factor: PH, physiology
Receptors, Cell Surface
Signal Transduction

*Smoke

Teichoic Acids: PD, pharmacology
Tobacco

Transcription, Genetic

src Homology Domains

CAS REGISTRY NO.: 117147-70-3 (amphiregulin); 56411-57-5 (lipoteichoic acid)
CHEMICAL NAME: 0 (Glycoproteins); 0 (Intercellular Signaling Peptides and
Proteins); 0 (Ligands); 0 (Lipopolysaccharides); 0
(Mucins); 0 (Receptors, Cell Surface); 0 (Teichoic Acids);
0 (diphtheria toxin receptor); EC 2.7.1.112 (Receptor,
Epidermal Growth Factor); EC 3.4.- (Endopeptidases); EC
3.4.23.- (secretase); EC 3.4.24 (Metalloendopeptidases); EC
3.4.24.- (TNF-alpha converting enzyme)

L136 ANSWER 4 OF 47 MEDLINE on STN
ACCESSION NUMBER: 2001246969 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11248147
TITLE: Rapid reduction of intracellular glutathione in human
bronchial epithelial cells exposed to occupational levels
of toluene diisocyanate.
AUTHOR: Lantz R C; Lemus R; Lange R W; Karol M H
CORPORATE SOURCE: Department of Cell Biology and Anatomy, The University of
Arizona, Tucson, Arizona 85724, USA..
lantz@email.arizona.edu
CONTRACT NUMBER: 05651 (NIEHS)
ES06694
SOURCE: Toxicological sciences : an official journal of the Society
of Toxicology, (2001 Apr) 60 (2) 348-55.
Journal code: 9805461. ISSN: 1096-6080.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200106
ENTRY DATE: Entered STN: 20010611
Last Updated on STN: 20010611
Entered Medline: 20010607

ABSTRACT:

Toluene diisocyanate (TDI) is a recognized chemical asthmogen, yet the mechanism of this toxicity and the molecular reactions involved have not been elucidated. We have previously shown that TDI vapor forms adducts with the apical surface of the respiratory epithelium, and that it colocalizes with ciliary tubulin. In vitro, we have shown rapid reaction of TDI with glutathione (GSH) and transfer of the bisGS-TDI adduct to a sulfhydryl-containing major histocompatibility complex peptide. This study sought to determine if intracellular GSH is altered following exposure to TDI. We used the dye CellTracker Green (chloromethylfluorescein, CMFDA) for detection of glutathione. One-day and 6-day air-liquid cultures of human bronchoepithelial cells (HBE) were exposed to 20-100 ppb TDI vapor for 5, 15, or 30 min. Cells were subsequently imaged using a confocal microscope. Both 1- and 6-day cultures showed a decrease in intensity of the thiol staining as a function of the TDI exposure dose. Doses as low as 20 ppb, the current permissible exposure limit (PEL) to TDI, resulted in rapid (within 5 min) decreases in fluorescence. The decreased fluorescence was not due to cytotoxicity or decrease in either esterase or glutathione-S-transferase activity, enzymes necessary for activation of the fluorescence of CMFDA. The decrease in glutathione levels was verified using another fluorescent label,

ThioGlo(TM) 1, and cell extracts. In addition, the mucus produced by 6-day air-liquid interface HBE cells in response to TDI exposure appeared to be protective, as HBE cells underlying mucus retained more fluorescence than did cells in the same cultures that were not covered with mucus. These results, along with previous data, strongly suggest that TDI enters pulmonary cells and reacts rapidly with intracellular GSH, and that this can occur at the current PEL of 20 ppb. This rapid reaction suggests the importance of cellular thiols in TDI-induced pulmonary disease.

CONTROLLED TERM: *Air Pollutants, Occupational: TO, toxicity
*Bronchi: DE, drug effects
Bronchi: ME, metabolism
Bronchi: PA, pathology
Cells, Cultured
Dose-Response Relationship, Drug
*Epithelial Cells: DE, drug effects
Epithelial Cells: ME, metabolism
Epithelial Cells: PA, pathology
Fluoresceins: ME, metabolism
Fluorescence
Fluorescent Dyes: ME, metabolism
Glutathione: DE, drug effects
*Glutathione: ME, metabolism
Humans
Microscopy, Confocal
Mucins: ME, metabolism
Research Support, U.S. Gov't, P.H.S.
Time Factors
*Toluene 2,4-Diisocyanate: TO, toxicity
CAS REGISTRY NO.: 136832-63-8 (5-chloromethylfluorescein); 584-84-9 (Toluene 2,4-Diisocyanate); 70-18-8 (Glutathione)
CHEMICAL NAME: 0 (Air Pollutants, Occupational); 0 (Fluoresceins); 0 (Fluorescent Dyes); 0 (Mucins)

L136 ANSWER 5 OF 47 MEDLINE on STN
ACCESSION NUMBER: 2000105580 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10637131
TITLE: Lung mucin production is stimulated by the air pollutant residual oil fly ash.
AUTHOR: Longphre M; Li D; Li J; Matovinovic E; Gallup M; Samet J M; Basbaum C B
CORPORATE SOURCE: Department of Anatomy and Cardiovascular Research
Institute, University of California, San Francisco 94143, USA.
CONTRACT NUMBER: P01HL24136 (NHLBI)
R01HL43762 (NHLBI)
SOURCE: Toxicology and applied pharmacology, (2000 Jan 15) 162 (2) 86-92.
Journal code: 0416575. ISSN: 0041-008X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200003
ENTRY DATE: Entered STN: 20000314
Last Updated on STN: 20000314
Entered Medline: 20000302

ABSTRACT:
Human and animal exposure to particulate air pollution is correlated with airway mucus hypersecretion and increased susceptibility to infection. Seeking clues to the mechanisms underlying this pathology, we examined the effect of

the particulate air pollutant residual oil fly ash (ROFA) on production of the major component of mucus, mucin, and the major antibacterial protein of the respiratory tract, lysozyme. We found that following in vitro exposure to ROFA, epithelial cells showed an increase in mucin (MUC5AC) and lysozyme (LYS) steady state mRNA. This upregulation was controlled at least partly at the level of transcription as shown by reporter assays. Experiments testing the ability of the major components of ROFA to mimic these effects showed that vanadium, a metal making up 18.8% by weight, accounted for the bulk of the response. A screen of signaling inhibitors showed that MUC5AC and LYS induction by ROFA are mediated by dissimilar signaling pathways, both of which are, however, phosphotyrosine dependent. Recognizing that the ROFA constituent vanadium is a potent tyrosine phosphatase inhibitor and that mucin induction by pathogens is phosphotyrosine dependent, we suggest that vanadium-containing air pollutants trigger disease-like conditions by unmasking phosphorylation-dependent pathogen resistance pathways.

Copyright 2000 Academic Press.

CONTROLLED TERM: *Air Pollutants, Environmental: TO, toxicity

Bronchi: CY, cytology

Bronchi: DE, drug effects

Cell Line

Cytoplasm: DE, drug effects

Cytoplasm: ME, metabolism

Cytoplasm: PH, physiology

Dose-Response Relationship, Drug

Epithelial Cells: DE, drug effects

*Fuel Oils: TO, toxicity

Humans

*Lung: DE, drug effects

*Lung: ME, metabolism

Lung Neoplasms

*Mucins: BI, biosynthesis

Particle Size

RNA, Messenger: ME, metabolism

Research Support, U.S. Gov't, P.H.S.

Signal Transduction: DE, drug effects

Transcription, Genetic: DE, drug effects

Tumor Cells, Cultured

*Vanadium: TO, toxicity

7440-62-2 (Vanadium)

CAS REGISTRY NO.:
CHEMICAL NAME: 0 (Air Pollutants, Environmental); 0 (Mucins); 0 (RNA, Messenger)

L136 ANSWER 6 OF 47

ACCESSION NUMBER:

DOCUMENT NUMBER:

TITLE:

AUTHOR:

CORPORATE SOURCE:

SOURCE:

PUB. COUNTRY:

DOCUMENT TYPE:

LANGUAGE:

MEDLINE on STN

1999036712 MEDLINE

PubMed ID: 9819294

Comparison of ciliary activity and inflammatory mediator release from bronchial epithelial cells of nonatopic nonasthmatic subjects and atopic asthmatic patients and the effect of diesel exhaust particles in vitro.

Bayram H; Devalia J L; Khair O A; Abdelaziz M M; Sapsford R J; Sagai M; Davies R J

Academic Department of Respiratory Medicine, St Bartholomew's and the Royal London School of Medicine and Dentistry, The London Chest Hospital, London E2 9JX, UK.

Journal of allergy and clinical immunology, (1998 Nov) 102 (5) 771-82.

Journal code: 1275002. ISSN: 0091-6749.

United States

Journal; Article; (JOURNAL ARTICLE)

English

Searched by Barb O'Bryen, STIC 2-2518

FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
 ENTRY MONTH: 199812
 ENTRY DATE: Entered STN: 19990115
 Last Updated on STN: 19990115
 Entered Medline: 19981209

ABSTRACT:

BACKGROUND: Recent studies have suggested that asthmatic patients may be more susceptible to the adverse effects of air pollutants, including diesel exhaust particles (DEP). The underlying mechanisms, however, are not clear. **METHODS:** We cultured bronchial epithelial cells from bronchial biopsy specimens of well-characterized groups of nonatopic, nonasthmatic individuals and atopic patients with mild asthma and compared the ciliary beat frequency (CBF) and release of IL-8, GM-CSF, regulated on activation, normal T-cell expressed and secreted (RANTES), and soluble intercellular adhesion molecule-1 (sICAM-1) from these cells both before and after exposure for 24 hours to 10 to 100 micrograms/mL DEP in vitro. **RESULTS:** The baseline CBF was not found to be significantly different in the bronchial epithelial cell cultures of nonasthmatic and asthmatic individuals. Incubation with DEP significantly attenuated the CBF of both the nonasthmatic and asthmatic bronchial epithelial cell cultures at all concentrations of DEP investigated and were maximal (55.5% and 45.2%, respectively) after incubation with 100 micrograms/mL DEP. The bronchial epithelial cell cultures of asthmatic patients constitutively released significantly greater amounts of IL-8, GM-CSF, and sICAM-1 than bronchial epithelial cell cultures of nonasthmatic subjects. The cultures of only asthmatic patients additionally released RANTES. Incubation of the asthmatic cultures with 10 micrograms/mL DEP significantly increased the release of IL-8 (from 102.0 to 167.8 pg/micrograms cellular protein; $P < .01$), GM-CSF (from 0.43 to 1.87 pg/micrograms cellular protein; $P < .01$), and sICAM-1 (from 14.7 to 38.1 pg/micrograms cellular protein; $P < .02$) after 24 hours. Incubation with 50 to 100 micrograms/mL DEP, however, significantly decreased the release of IL-8 and RANTES from these cultures. In contrast, only the higher concentrations of 50 to 100 micrograms/mL DEP significantly increased release of IL-8 (from 37.9 to 71.5 pg/micrograms cellular protein; $P < .05$) and GM-CSF (from 0.06 to 0.34 pg/micrograms cellular protein; $P < .05$) from the bronchial epithelial cells of nonasthmatic individuals. **CONCLUSIONS:** These results suggest that bronchial epithelial cells of asthmatic subjects are different from bronchial epithelial cells of nonasthmatic subjects with regard to the amounts and types of proinflammatory mediators they can release and that the increased sensitivity of bronchial epithelial cells of asthmatic subjects to DEP may possibly result in exacerbation of their disease symptoms.

CONTROLLED TERM: Check Tags: Comparative Study; Female; Male
 Adult
 *Asthma: ME, metabolism
 *Asthma: PP, physiopathology
 *Bronchi: CY, cytology
 Cells, Cultured
 *Cilia: PH, physiology
 Epithelial Cells: CY, cytology
 Humans
 *Hypersensitivity, Immediate: ME, metabolism
 *Hypersensitivity, Immediate: PP, physiopathology
 *Inflammation Mediators: ME, metabolism
 Lung Diseases, Obstructive: ME, metabolism
 Lung Diseases, Obstructive: PP, physiopathology
 Research Support, Non-U.S. Gov't
 *Vehicle Emissions: AE, adverse effects
 CHEMICAL NAME: 0 (Inflammation Mediators); 0 (Vehicle Emissions)

L136 ANSWER 7 OF 47 MEDLINE on STN
 ACCESSION NUMBER: 1998158681 MEDLINE

DOCUMENT NUMBER: PubMed ID: 9490663
TITLE: The effect of diesel exhaust particles on cell function and release of inflammatory mediators from human bronchial epithelial cells in vitro.
AUTHOR: Bayram H; Devalia J L; Sapsford R J; Ohtoshi T; Miyabara Y; Sagai M; Davies R J
CORPORATE SOURCE: Academic Department of Respiratory Medicine, St. Bartholomew's and the Royal London School of Medicine and Dentistry, The London Chest Hospital, London, United Kingdom.
SOURCE: American journal of respiratory cell and molecular biology, (1998 Mar) 18 (3) 441-8.
Journal code: 8917225. ISSN: 1044-1549.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199803
ENTRY DATE: Entered STN: 19980410
Last Updated on STN: 19980410
Entered Medline: 19980331

ABSTRACT:

Animal studies have reported that diesel exhaust particles (DEP), which constitute an important fraction of particulate air pollution, lead to inflammation and/or damage of the airways. To investigate the mechanisms underlying DEP-induced airway disease in humans, we have cultured human bronchial epithelial cells (HBEC) from surgically obtained bronchial explants and investigated the effects of purified DEP on the permeability and ciliary beat frequency (CBF) of HBEC, and on the release of inflammatory mediators from these cells. Exposure to 10-100 microg/ml DEP and a filtered solution of 50 microg/ml DEP significantly increased the electrical resistance of the cultures, reaching a maximum of 200% over baseline after 6 h incubation with 100 microg/ml DEP. In contrast, movement of ¹⁴C-labeled bovine serum albumin across cell cultures was not significantly altered by incubation of HBEC with DEP. Exposure to 50 microg/ml DEP, filtered DEP solution, and 100 microg/ml DEP significantly attenuated the CBF of these cells by 51%, 33%, and 73%, respectively, from baseline after 24 h incubation. Similarly, 50 microg/ml DEP, filtered DEP solution, and 100 microg/ml DEP significantly increased the release of interleukin-8 from 12.9 pg/microg cellular protein to 41.6, 114.9, and 44.3 pg/microg cellular protein, respectively, after 24 h incubation. The release of granulocyte-macrophage colony stimulating factor (GM-CSF) and soluble intercellular adhesion molecule-1 (sICAM-1) was also significantly increased after exposure for 24 h to 50 microg/ml DEP (GM-CSF from 0.033 pg/microg cellular protein to 0.056 pg/mug cellular protein and sICAM-1 from 7.2 pg/microg cellular protein to 12.5 pg/microg cellular protein). These results suggest that exposure of HBEC to DEP may lead to adverse functional changes and release of proinflammatory mediators from these cells, and that these effects may influence the development of airway disease.

CONTROLLED TERM: Adult

Air Pollutants, Environmental: CH, chemistry
*Air Pollutants, Environmental: TO, toxicity
Bronchi: CY, cytology
*Bronchi: IM, immunology
Cell Membrane Permeability: DE, drug effects
Cells, Cultured
Cilia: DE, drug effects
Epithelial Cells: CY, cytology
*Epithelial Cells: IM, immunology
Granulocyte-Macrophage Colony-Stimulating Factor: SE, secretion

Humans

*Inflammation Mediators: ME, metabolism

Intercellular Adhesion Molecule-1: BI, biosynthesis

Interleukin-8: SE, secretion

Lung Diseases: ET, etiology

Middle Aged

Polycyclic Compounds: TO, toxicity

Research Support, Non-U.S. Gov't

*Vehicle Emissions: TO, toxicity

CAS REGISTRY NO.: 126547-89-5 (Intercellular Adhesion Molecule-1); 83869-56-1
(Granulocyte-Macrophage Colony-Stimulating Factor)
CHEMICAL NAME: 0 (Air Pollutants, Environmental); 0 (Inflammation
Mediators); 0 (Interleukin-8); 0 (Polycyclic Compounds); 0
(Vehicle Emissions)

L136 ANSWER 8 OF 47

MEDLINE on STN

ACCESSION NUMBER: 93310724 MEDLINE

DOCUMENT NUMBER: PubMed ID: 8322244

TITLE: Effect of azelastine on sulphur dioxide induced impairment
of ciliary motility in airway epithelium.

AUTHOR: Tamaoki J; Chiyotani A; Sakai N; Takeyama K; Konno K

CORPORATE SOURCE: First Department of Medicine, Tokyo Women's Medical
College, Japan.SOURCE: Thorax, (1993 May) 48 (5) 542-6.
Journal code: 0417353. ISSN: 0040-6376.

PUB. COUNTRY: ENGLAND: United Kingdom

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199308

ENTRY DATE: Entered STN: 19930813

Last Updated on STN: 19930813

Entered Medline: 19930803

ABSTRACT:

OBJECTIVE--The effect of azelastine on airway mucociliary transport function was studied by measuring ciliary motility of human bronchial epithelium in vitro with a photoelectric method. METHOD--Bronchial epithelial cells were obtained by fiberoptic bronchoscopy, mounted in a Rose chamber, and perfused with Krebs-Henseleit solution. The preparations were placed on a microscope stage equipped with an illuminator, and the variations of light intensity caused by ciliary beating were detected by a photometer. RESULTS--The addition of azelastine to the perfusate increased ciliary beat frequency (CBF) in a dose dependent manner without ciliary discoordination. The mean (SE) maximal increase from the baseline value and the concentration required to produce a half maximal effect were 27.0 (4.2)% and 9.2×10^{-6} mol/l, respectively. Exposure of the cells to the perfusate containing 3 ppm sulphur dioxide rapidly decreased CBF by 59.2 (5.0)%, and was accompanied by a reduction in intracellular cyclic AMP levels from 38.1 (4.3) to 10.1 (2.4) pmol/mg protein. This effect was prevented by pretreatment of cells with azelastine in a dose dependent manner. CONCLUSIONS--Azelastine not only stimulates ciliary motility of airway epithelium and hence mucociliary transport function, but may also protect against sulphur dioxide induced ciliary dysfunction, probably by inhibiting intracellular cyclic AMP loss.

CONTROLLED TERM:

*Bronchi: DE, drug effects

*Bronchodilator Agents: PD, pharmacology
Cells, Cultured

*Cilia: DE, drug effects

Epithelial Cells

Epithelium: DE, drug effects

Humans

Searched by Barb O'Bryen, STIC 2-2518

Mucociliary Clearance: DE, drug effects
 Photometry
 *Phthalazines: PD, pharmacology
 Research Support, Non-U.S. Gov't
 Stimulation, Chemical
 Sulfur Dioxide: PD, pharmacology
 CAS REGISTRY NO.: 58581-89-8 (azelastine); 7446-09-5 (Sulfur Dioxide)
 CHEMICAL NAME: 0 (Bronchodilator Agents); 0 (Phthalazines)

L136 ANSWER 9 OF 47 MEDLINE on STN
 ACCESSION NUMBER: 89007987 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 3170450
 TITLE: Ciliated cells in vitamin A-deprived cultured hamster
 tracheal epithelium do divide.
 AUTHOR: Rutten A A; Beems R B; Wilmer J W; Feron V J
 CORPORATE SOURCE: TNO-CIVO Toxicology and Nutrition Institute, Zeist,
 Netherlands.
 SOURCE: In vitro cellular & developmental biology : journal of the
 Tissue Culture Association, (1988 Sep) 24 (9) 931-5.
 Journal code: 8506951. ISSN: 0883-8364.
 PUB. COUNTRY: United States
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 198811
 ENTRY DATE: Entered STN: 19900308
 Last Updated on STN: 19900308
 Entered Medline: 19881110

ABSTRACT:

The pseudostratified tracheal epithelium, composed of a heterogeneous phenotypically varying cell population, was studied with respect to the in vitro cell proliferative activity of differentiated epithelial cells. Ciliated tracheal epithelial cells so far have been considered to be terminally differentiated, nonproliferating cells. Tracheal organ cultures obtained from vitamin A-deprived Syrian Golden hamsters were cultured in a vitamin A-deficient, serum-free, hormone-supplemented medium. In vitamin A-deprived tracheal epithelium treated with physiologically active all-trans retinol and low cigarette-smoke condensate concentrations it is possible to stimulate the cell proliferation of both basal and columnar cells. Therefore, the probability of finding proliferating columnar cells was increased compared with the in vivo and the vitamin A-deprived situation in which cell proliferative activity is relatively low. In the presence of cigarette-smoke condensate in a noncytotoxic concentration, basal, small mucous granule, ciliated, and indifferent tracheal epithelial cells incorporated [methyl-3H]-thymidine into the DNA during the S phase. The finding that ciliated cells were labeled was supported by serial sections showing the same labeled ciliated cell in two section planes separated by 2 to 3 micron, without labeled epithelial cells next to the ciliated cell. Furthermore, a ciliated tracheal epithelial cell incorporating [methyl-3H]thymidine into DNA was also seen in tracheal cultures of vitamin A-deprived hamsters treated with all-trans retinol in a physiologic concentration.

CONTROLLED TERM:

Animals
 Autoradiography
 Cell Differentiation: DE, drug effects
 Cell Division: DE, drug effects
 Cells, Cultured
 Cilia
 Epithelial Cells
 Epithelium: DE, drug effects
 Hamsters

Mesocricetus
 Plants, Toxic
 Research Support, Non-U.S. Gov't
 Smoke
 Tobacco
 *Trachea: CY, cytology
 Trachea: DE, drug effects
 Vitamin A: PD; pharmacology
 *Vitamin A Deficiency: PA, pathology
 CAS REGISTRY NO.: 11103-57-4 (Vitamin A)

L136 ANSWER 10 OF 47 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 1
 2004:14199 CAPLUS

ACCESSION NUMBER: 140:265914

DOCUMENT NUMBER: 140:265914

TITLE: Responses of cultured human airway epithelial cells treated with diesel exhaust extracts will vary with the engine load

AUTHOR(S): Madden, Michael C.; Dailey, Lisa A.; Stonehuerner, Jacqueline G.; Harris, D. Bruce

CORPORATE SOURCE: Natl. Health and Environmental Effects Research Laboratory, ORD, U.S. Environmental Protection Agency, Research Triangle Park, NC, USA

SOURCE: Journal of Toxicology and Environmental Health, Part A (2003), 66(24), 2281-2297
 CODEN: JTEHF8; ISSN: 1528-7394

PUBLISHER: Taylor & Francis, Inc.

DOCUMENT TYPE: Journal

LANGUAGE: English

ED Entered STN: 09 Jan 2004

AB Epidemiol. evidence suggests that increased morbidity and mortality are associated with the concns. of ambient air particulate matter (PM). Many sources contribute to the particulate fraction of ambient pollution, including diesel exhaust particulates (DEP). Diesel exhaust also contributes gas-phase pollutants to the atmospheric, and gaseous copollutants

may influence the toxicity of PM. The composition of diesel exhaust varies greatly depending on the engine load conditions as well as other factors. To determine whether different diesel exhaust composition can affect lung cell responses, the effects of diesel exhaust exts. derived from different engine loads were examined on normal human bronchial epithelial cells (NHBE) in vitro. Diesel exhaust was collected into chilled impingers containing phosphate-buffered saline (PBS). Cultured NHBE cells were treated with 0 to 500 µg/well of extract from .apprx.0% engine load (termed low load or LL) or extract from .apprx.75% engine load (termed high load or HL) for 24h. The HL extract was cytotoxic at 500 µg compared to controls as measured by 51Cr release. Production of the neutrophil chemotaxin interleukin 8 (IL-8) was decreased 4.7-fold in cells treated with 500 µgLL extract, whereas cells treated with 500 µg HL extract showed a 2.4-fold increase in IL-8 release. Production of the inflammatory and immune system mediator prostaglandin E2 (PGE2) was increased up to 2.5-fold in cells treated with HL extract, but unchanged with other treatments. Melittin stimulation of cells showed that the LL extract had an inhibitory effect on PGE2 release at 500 µg. Differences in carbonyl content of the exts. were found by high-performance liquid chromatog./mass spectrometry (HPLC/MS), with the HL extract having more intermediate size carbonyls (i.e., with six to nine carbons). The data suggest that the response of NHBE cells to treatment with diesel exhaust will vary depending on the constituent components of

- the exhaust.
- CC 4-3 (Toxicology)
Section cross-reference(s): 59
- IT Aldehydes, biological studies
RL: ADV (Adverse effect, including toxicity); POL (Pollutant); BIOL (Biological study); OCCU (Occurrence)
(C2-C12; responses of **cultured** human airway epithelial cells treated with diesel exhaust exts. will vary with engine load)
- IT Exhaust gases (engine)
(diesel, extract; responses of **cultured** human airway epithelial cells treated with diesel exhaust exts. will vary with engine load)
- IT Respiratory system
(epithelium; responses of **cultured** human airway epithelial cells treated with diesel exhaust exts. will vary with engine load)
- IT Air pollution
(exhaust; responses of **cultured** human airway epithelial cells treated with diesel exhaust exts. will vary with engine load)
- IT Epithelium
(respiratory tract; responses of **cultured** human airway epithelial cells treated with diesel exhaust exts. will vary with engine load)
- IT Human
(responses of **cultured** human airway epithelial cells treated with diesel exhaust exts. will vary with engine load)
- IT Interleukin 8
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(responses of **cultured** human airway epithelial cells treated with diesel exhaust exts. will vary with engine load)
- IT 363-24-6, Prostaglandin E2 329900-75-6, Cyclooxygenase-2
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(responses of **cultured** human airway epithelial cells treated with diesel exhaust exts. will vary with engine load)
- REFERENCE COUNT: 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L136 ANSWER 11 OF 47 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 5
ACCESSION NUMBER: 1999:302795 CAPLUS
DOCUMENT NUMBER: 131:69365
TITLE: Evaluation of two in vitro ciliated epithelial systems, dog trachea and frog palate, for potential as screens for sensory irritation
AUTHOR(S): Swann, J. M.; Kennedy, J. R.; Schultz, T. W.
CORPORATE SOURCE: Department of Biochemistry, Cellular, and Molecular Biology, The University of Tennessee, Knoxville, TN, 37996-4500, USA
SOURCE: In Vitro & Molecular Toxicology (1999), 12(1), 17-32
CODEN: IVMTFJ; ISSN: 1097-9336
PUBLISHER: Mary Ann Liebert, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 18 May 1999
AB Two aquatic-based in vitro ciliated epithelial systems, dog trachea and frog palate, have been evaluated as potential screens for sensory irritation of inhaled chems. Initially, the effects of a series of nonpolar narcotic agents (i.e., alcs. and ketones) were assessed by monitoring ciliary beat frequency under standard (static, 5-min exposure) conditions with the concentration (M) eliciting a 50% reduction in

ciliary beat frequency (i.e., EC50) being determined. These data along with 1-octanol per water partition coefficient (log KOW) were used to formulate baseline predictive models. The minimal toxicity model for the canine trachea (ct) system is $\log 1/ctEC50 = 0.79 (\log KOW) - 0.002$; $n = 7$, $r^2 = 0.987$, $s = 0.15$, $F = 373$, $Pr > F = 0.0001$. The frog palate (fp) baseline toxicity equation is $\log 1/fpEC50 = 0.80 (\log KOW) - 0.12$; $n = 7$, $r^2 = 0.995$, $s = 0.09$, $F = 1112$, $Pr > F = 0.0001$. Prevalidation of both in vitro systems were undertaken by regression analyses with in vivo mouse inhalation toxicity data. The slopes of both analyses are not statistically different than 1.0. The intercepts reveal the in vivo endpoint to be tenfold more sensitive. Also, a set of bioreactive chems. was tested. Mechanisms of toxicity represented by this set of chems. included three specific electrophilic or nucleophilic mechanisms, weak acid respiratory uncoupling, and reversible and irreversible acetylcholinesterase inhibition. Although the relative ranking varied between the two in vitro systems, all bioreactive chems. were to have toxicity greater than twice that predicted by the baseline models. Based on these preliminary data, we feel that such a **ciliary beat frequency** in vitro system would provide valuable hazard information about potential sensory irritation of inhaled chems.

CC 4-1 (Toxicology)

IT Chemicals

Palate

Simulation and Modeling, biological

Toxicity

Trachea (anatomical)

(evaluation of two in vitro **ciliated epithelial**

systems, dog trachea and frog palate, for potential as screens for sensory irritation)

IT Alcohols, biological studies

Ketones, biological studies

RL: ADV (Adverse effect, including toxicity); PRP (Properties); BIOL

(Biological study)

(evaluation of two in vitro **ciliated epithelial**

systems, dog trachea and frog palate, for potential as screens for sensory irritation)

IT **Toxicity**(inhalation; evaluation of two in vitro **ciliated epithelial** systems, dog trachea and frog palate, for potential as screens for sensory irritation)

IT 63-25-2, Carbaryl 64-17-5, Ethanol, biological studies 71-41-0,

1-Pentanol, biological studies 78-93-3, 2-Butanone, biological studies

100-44-7, Benzyl chloride, biological studies 107-87-9, 2-Pentanone

111-70-6, 1-Heptanol 111-71-7, Heptanal 111-87-5, 1-Octanol,

biological studies 112-12-9, 2-Undecanone 121-75-5, Malathion

534-52-1, 4,6-Dinitro-o-cresol 693-54-9, 2-Decanone 2548-87-0

RL: ADV (Adverse effect, including toxicity); PRP (Properties); BIOL

(Biological study)

(evaluation of two in vitro **ciliated epithelial**

systems, dog trachea and frog palate, for potential as screens for sensory irritation)

REFERENCE COUNT:

37

THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L136 ANSWER 12 OF 47 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 7

ACCESSION NUMBER: 1996:88536 CAPLUS

DOCUMENT NUMBER: 124:138291

TITLE: The effects of the organophosphorus insecticides

Dursban and Lorsban on the **ciliated****epithelium** of the frog plate in vitro

Searched by Barb O'Bryen, STIC 2-2518

AUTHOR(S): Swann, J. M.; Schultz, T. W.; Kennedy, J. R.
 CORPORATE SOURCE: Univ. Tennessee, Coll. Veterinary Med., Knoxville, TN,
 37996, USA
 SOURCE: Archives of Environmental Contamination and Toxicology
 (1996), 30(2), 188-94
 CODEN: AECTCV; ISSN: 0090-4341
 PUBLISHER: Springer
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 ED Entered STN: 10 Feb 1996
 AB The ciliotoxic potential of the organophosphorous insecticides Dursban and Lorsban, their active ingredient, chlorpyrifos, and their carrier ingredients (Blanks) were assessed. Since chlorpyrifos inhibits acetylcholinesterase, the acetylcholine-innervated ciliated epithelial cultures of frog palate were used as the model. All compds. caused a decrease in frequency of **ciliary beat** over time. EC50 values followed the same order as the time to inhibition. The orders were Lorsban > Dursban > chlorpyrifos, and Lorsban > Dursban .apprx. Lorsban Blank > Dursban Blank. Stimulation of **ciliary beating** occurred immediately after exposure to all compds., followed by inhibition. Dursban, Lorsban, and both Blanks elicited stimulatory effects in the presence of atropine. Atropine only blocked the initial stimulatory response with chlorpyrifos. In addition to chlorpyrifos, some component(s) of the inert ingredients were initially stimulatory but ultimately inhibitory to **ciliary beating** in the frog palate model. All compds. caused mitochondrial damage, including swelling, disruption of cristae, and loss of matrix.
 CC 4-4 (Toxicology)
 ST organophosphorous insecticide **ciliated epithelium**
 palate frog; dursban lorsban chlorpyrifos **ciliated**
epithelium frog; Rana organophosphorous insecticide
ciliated epithelium
 IT Cell morphology
 Insecticides
 Mitochondria
 Rana pipiens
 Toxicity
 (toxicity of organophosphorus insecticides Dursban and Lorsban on the
ciliated epithelium of the frog plate in vitro)
 IT **Epithelium**
 (**ciliated**, toxicity of organophosphorus insecticides Dursban
 and Lorsban on the **ciliated epithelium** of the frog
 plate in vitro)
 IT Palate
 (**epithelium**, toxicity of organophosphorus insecticides
 Dursban and Lorsban on the **ciliated epithelium** of
 the frog plate in vitro)
 IT Organic compounds, biological studies
 RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
 (phosphorus-containing, toxicity of organophosphorus insecticides Dursban
 and Lorsban on the **ciliated epithelium** of the frog
 plate in vitro)
 IT 2921-88-2, Dursban
 RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
 (toxicity of organophosphorus insecticides Dursban and Lorsban on the
ciliated epithelium of the frog plate in vitro)
 IT 51-55-8, Atropine, biological studies
 RL: BAC (Biological activity or effector, except adverse); BSU (Biological
 study, unclassified); BIOL (Biological study)
 (toxicity of organophosphorus insecticides Dursban and Lorsban on the

ciliated epithelium of the frog plate in vitro)

L136 ANSWER 13 OF 47 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2005:968536 CAPLUS

DOCUMENT NUMBER: 143:481862

TITLE: Influence of seasons and sampling strategy on assessment of bioaerosols in sewage treatment plants in Switzerland

AUTHOR(S): Oppliger, Anne; Hilfiger, Silvia; Duc, Trinh Vu
CORPORATE SOURCE: Institute of Occupational Health Sciences, Lausanne, CH-1015, Switz.

SOURCE: Annals of Occupational Hygiene (2005), 49(5), 393-400
CODEN: AOHYA3; ISSN: 0003-4878

PUBLISHER: Oxford University Press

DOCUMENT TYPE: Journal

LANGUAGE: English

ED Entered STN: 06 Sep 2005

AB An assessment of sewage workers' exposure to airborne cultivable bacteria, fungi and inhaled endotoxins was performed at 11 sewage treatment plants. We sampled the enclosed and unenclosed treatment areas in each plant and evaluated the influence of seasons (summer and winter) on bioaerosol levels. We also measured personal exposure to endotoxins of workers during special operation where a higher risk of bioaerosol inhalation was assumed. Results show that only fungi are present in significantly higher concns. in summer than in winter (2331 ± 858 vs. 329 ± 95 CFU m⁻³). We also found that there are significantly more bacteria in the enclosed area, near the particle grids for incoming water, than in the unenclosed area near the aeration basins (9455 ± 2661 vs. 2435 ± 985 CFU m⁻³ in summer and 11081 ± 2299 vs. 2002 ± 839 CFU m⁻³ in winter). All bioaerosols were frequently above the recommended values of occupational exposure. Workers carrying out special tasks such as cleaning tanks were exposed to very high levels of endotoxins (up to 500 EU m⁻³) compared to routine work. The species composition and concentration of airborne Gram-neg. bacteria were also studied. A broad spectrum of different species within the Pseudomonadaceae and the Enterobacteriaceae families were predominant in nearly all plants investigated.

CC 59-5 (Air Pollution and Industrial Hygiene)
Section cross-reference(s): 4, 10

IT Acinetobacter baumannii
Acinetobacter junii
Aeromonas hydrophila

Air pollution

Burkholderia cepacia
Chromobacterium violaceum
Chryseomonas luteola
Comamonas acidovorans
Enterobacter aerogenes
Enterobacter cloacae
Enterobacteriaceae
Escherichia coli
Fungi
Human
Industrial hygiene
Klebsiella oxytoca
Klebsiella pneumoniae
Occupational health hazard
Ochrobactrum anthropi
Pantoea agglomerans
Pseudomonadaceae
Pseudomonas aeruginosa

Pseudomonas indologenes
 Pseudomonas oryzihabitans
 Pseudomonas pseudoalcaligenes
 Pseudomonas putida
 Ralstonia pickettii
 Raoultella terrigena
 Salmonella choleraesuis arizonae

Sampling

Serratia ficaria
 Serratia proteamaculans proteamaculans
 Temperature
 Wastewater treatment

(bioaerosols occupational exposure assessment in different season, for different work sites and during special tasks in wastewater treatment plant in Switzerland)

IT Eubacteria

(culturable; bioaerosols occupational exposure assessment in different season, for different work sites and during special tasks in wastewater treatment plant in Switzerland)

REFERENCE COUNT: 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L136 ANSWER 14 OF 47 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2005:505432 CAPLUS

TITLE: Integrating measurements of radon and thoron and their deposition fractions in the respiratory tract

AUTHOR(S): Zhuo, W.; Tokonami, S.; Yonehara, H.; Yamada, Y.

CORPORATE SOURCE: Radon Research Group, National Institute of

Radiological Sciences, Inage, Chiba, 263-8555, Japan

SOURCE: Radioactivity in the Environment (2005), 7 (Natural Radiation Environment VII), 352-360

CODEN: REANCK

PUBLISHER: Elsevier Ltd.

DOCUMENT TYPE: Journal

LANGUAGE: English

ED Entered STN: 14 Jun 2005

AB For simultaneous measurements of indoor ^{222}Rn and ^{220}Rn and estimation of their deposition fractions in the respiratory tract, a new type of passive integrating ^{222}Rn and ^{220}Rn monitor and a portable bronchial dosimeter were developed. The passive ^{222}Rn and ^{220}Rn monitor was rebuilt from a com. available passive ^{222}Rn monitor. Besides its simple construction, the volume and weight of the new monitor are only 110 cm³ and 20 g. Calibration factors of ^{222}Rn and ^{220}Rn for the new monitor were systematically studied through calibration expts. The results indicated that indoor ^{220}Rn could be discriminated from ^{222}Rn by using the new passive monitor. The bronchial dosimeter consists of three sets of progeny integrating sampling units (PISUs) with different configurations of sampling heads. Multiple metal screens are used to mimic the penetrating and deposition behavior of $^{222}\text{Rn}/^{220}\text{Rn}$ progeny in the nasal and tracheo-bronchial (T-B) regions of the human respiratory tract. The potential alpha energy concns. (PAEC) of $^{222}\text{Rn}/^{220}\text{Rn}$ progeny are directly measured with the allyl diglycol carbonate (CR-39) detectors inside the PISUs. The deposition fractions of ^{222}Rn and ^{220}Rn progeny in the T-B region were measured, with avs. of 4.5 and 4.0% for ordinary room conditions, in general agreement with other reported values. Both the new monitor and device are simple and compact as well as of low cost, and they are considered to be practical for large-scale and long-term surveys of indoor ^{222}Rn and ^{220}Rn .

CC 9 (Biochemical Methods)

IT INDEXING IN PROGRESS

- IT **Respiratory system**
(epithelium; integrated measurements of radon and thoron and their deposition fractions in the **respiratory** tract).
- IT **Biosensors**
(integrated measurements of radon and thoron and their deposition fractions in the respiratory tract using a bronchial dosimeter)
- IT **Epithelium**
(**respiratory** tract; integrated measurements of radon and thoron and their deposition fractions in the **respiratory** tract)

REFERENCE COUNT: 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L136 ANSWER 15 OF 47 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2005:337517 CAPLUS

DOCUMENT NUMBER: 143:119370

TITLE: Pro-inflammatory effects of Dunkerque city air pollution particulate matter 2.5 in human epithelial lung cells (L132) in **culture**

AUTHOR(S): Dagher, Zeina; Garcon, Guillaume; Gosset, Pierre; Ledoux, Frederic; Surpateanu, Georgiana; Courcot, Dominique; Aboukais, Antoine; Puskaric, Emile; Shirali, Pirouz

CORPORATE SOURCE: Laboratoire de Recherche en Toxicologie Industrielle et Environnementale, Maison de la Recherche en Environnement Industriel de Dunkerque 2, Dunkerque, 59140, Fr.

SOURCE: Journal of Applied Toxicology (2005), 25(2), 166-175
CODEN: JJATDK; ISSN: 0260-437X

PUBLISHER: John Wiley & Sons Ltd.

DOCUMENT TYPE: Journal

LANGUAGE: English

ED Entered STN: 20 Apr 2005

AB Exposure to urban airborne particulate matter (PM) has been associated with adverse health effects. The majority of research articles published on air pollution PM relate to PM₁₀. However, increasing emphasis and stringent regulations have been placed on PM_{2.5}. The mechanisms for PM-induced adverse health effects are not well understood, but inflammation seems to be of importance. We focused our attention also on the capacity of air pollution PM_{2.5} to induce cytotoxic and inflammatory responses in human epithelial lung cells (L132) in culture. Particulate matter was collected in Dunkerque, a French seaside city characterized by the proximity of industrial activity and heavy motor vehicle traffic. Size distribution results showed that the cumulative frequency of PM_{2.5} was 92.15% and their sp. surface area was 1 m² g⁻¹. Inorg. and organic chems. usually associated with the natural environment but also so-called anthropogenic elements were found in PM, suggesting that much of the PM was derived from wind-borne dust from the industrial complex and the heavy diesel motor vehicle. We observed PM concentration-dependent cytotoxic effects in L132 cells (LC₁₀ = 18.84 µg PM ml⁻¹; LC₅₀ = 75.36 µg PM ml⁻¹). We showed that exposure to Dunkerque City's PM_{2.5} induced significant increases (in a concentration- and time-dependent manner) in protein secretion and/or gene expression of inflammatory cytokines (i.e. TNF-α, IL-1β, IL-8, GM-CSF, IL-6, TGF-β1). We hypothesized also that the occurrence of the acute inflammatory response might rely on the capacity of such air pollutants to generate oxidative species, which have been implicated in the stringent regulation of the cytokine network. Hence, we suggest that the development of inflammatory effects that worsen over time stems from the cytotoxicity in Dunkerque City's PM_{2.5}-exposed

CC L132 cells in culture.
59-2 (Air Pollution and Industrial Hygiene)
Section cross-reference(s): 4

IT Lung
(epithelium; pro-inflammatory effects of Dunkerque city air pollution particulate matter 2.5 in human epithelial lung cells (L132) in culture)

IT Waste gases
(industrial; pro-inflammatory effects of Dunkerque city air pollution particulate matter 2.5 in human epithelial lung cells (L132) in culture)

IT Respiratory system, disease
(inflammation; pro-inflammatory effects of Dunkerque city air pollution particulate matter 2.5 in human epithelial lung cells (L132) in culture)

IT Air pollution
(particulate; pro-inflammatory effects of dunkerque city air pollution particulate matter 2.5 in human epithelial lung cells (L132) in culture)

IT Cytotoxicity
Exhaust gases (engine)
Human
(pro-inflammatory effects of Dunkerque city air pollution particulate matter 2.5 in human epithelial lung cells (L132) in culture)

IT Interleukin 1 β
Interleukin 6
Interleukin 8
Tumor necrosis factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(pro-inflammatory effects of Dunkerque city air pollution particulate matter 2.5 in human epithelial lung cells (L132) in culture)

IT Health hazard
(pro-inflammatory effects of dunkerque city air pollution particulate matter 2.5 in human epithelial lung cells (L132) in culture)

IT Epithelium
(pulmonary; pro-inflammatory effects of Dunkerque city air pollution particulate matter 2.5 in human epithelial lung cells (L132) in culture)

IT Inflammation
(respiratory tract; pro-inflammatory effects of Dunkerque city air pollution particulate matter 2.5 in human epithelial lung cells (L132) in culture)

IT Airborne particles
(urban; pro-inflammatory effects of dunkerque city air pollution particulate matter 2.5 in human epithelial lung cells (L132) in culture)

IT Transforming growth factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(β 1-; pro-inflammatory effects of Dunkerque city air pollution particulate matter 2.5 in human epithelial lung cells (L132) in culture)

IT 83869-56-1, GM-CSF,
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(pro-inflammatory effects of Dunkerque city air pollution particulate matter 2.5 in human epithelial lung cells (L132) in culture)

REFERENCE COUNT: 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L136 ANSWER 16 OF 47 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2004:100900 CAPLUS

Searched by Barb O'Bryen, STIC 2-2518

DOCUMENT NUMBER: 140:132674
 TITLE: Real-time detection method and system for identifying individual aerosol particles
 INVENTOR(S): Gard, Eric Evan; Fergenson, David Philip
 PATENT ASSIGNEE(S): The Regents of the University of California, USA
 SOURCE: U.S. Pat. Appl. Publ., 16 pp.
 CODEN: USXXCO
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 2
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004024539	A1	20040205	US 2002-280608	20021024
US 6959248	B2	20051025		
US 2005073683	A1	20050407	US 2004-916737	20040811
			US 2001-335598P	P 20011025
			US 2002-280608	A2 20021024
			US 2003-494442P	P 20030811

PRIORITY APPLN. INFO.:

ED Entered STN: 08 Feb 2004

AB A method and system of identifying individual aerosol particles in real time. Sample aerosol particles are compared against and identified with substantially matching known particle types by producing pos. and neg. test spectra of an individual aerosol particle using a bipolar single particle mass spectrometer. Each test spectrum is compared to spectra of the same resp. polarity in a database of predetd. pos. and neg. spectra for known particle types and a set of substantially matching spectra is obtained. Finally the identity of the individual aerosol particle is determined from the set of substantially matching spectra by determining a best matching one of the known particle types having both a substantially matching pos. spectrum and a substantially matching neg. spectrum associated with the best matching known particle type.

IC ICM G06F019-00
 ICS G01N031-00

INCL 702028000

CC 59-2 (Air Pollution and Industrial Hygiene)
 Section cross-reference(s): 4, 10

IT **Airborne particles**
 (aerosols, anal. of; real-time detection method and system for identifying individual aerosol particles using bipolar single particle mass spectrometry)

IT **Culture media**
 (inclusion in sample; real-time detection method and system for identifying individual aerosol particles using bipolar single particle mass spectrometry)

IT **Air pollution**
 (particulate, aerosol, anal. of; real-time detection method and system for identifying individual aerosol particles using bipolar single particle mass spectrometry)

IT **Alarm devices**
Biological warfare agents
Chemical warfare agents
Sampling

Time-of-flight mass spectrometry
 (real-time detection method and system for identifying individual aerosol particles using bipolar single particle mass spectrometry)

L136 ANSWER 17 OF 47 CAPLUS COPYRIGHT 2005 ACS on STN
 ACCESSION NUMBER: 2003:913421 CAPLUS

Searched by Barb O'Bryen, STIC 2-2518

DOCUMENT NUMBER: 139:368744
 TITLE: Real-time detection method and system for identifying individual aerosol particles
 INVENTOR(S): Gard, Eric E.; Fergenson, David P.
 PATENT ASSIGNEE(S): The Regents of the University of California, USA
 SOURCE: PCT Int. Appl., 34 pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003096375	A1	20031120	WO 2002-US34291	20021025
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
			US 2001-280608	A 20011025

PRIORITY APPLN. INFO.:

ED Entered STN: 21 Nov 2003

AB A method and system of identifying individual aerosol particles in real time. Sample aerosol particles are compared against and identified with substantially matching known particle types by producing pos. and neg. test spectra of an individual aerosol particle using a bipolar single particle mass spectrometer. Each test spectrum is compared to spectra of the same resp. polarity in a database of predetd. pos. and neg. spectra for known particle types and a set of substantially matching spectra is obtained. Finally the identity of the individual aerosol particle is determined from the set of substantially matching spectra by determining a best matching one of the known particle types having both a substantially matching pos. spectrum and a substantially matching neg. spectrum associated with the best matching known particle type.

IC ICM H01J049-40

ICS H01J049-04; H01J049-16

CC 59-2 (Air Pollution and Industrial Hygiene)

Section cross-reference(s): 4, 10

IT Airborne particles

(aerosols, anal. of; real-time detection method and system for identifying individual aerosol particles using bipolar single particle mass spectrometry)

IT Culture media

(inclusion in sample; real-time detection method and system for identifying individual aerosol particles using bipolar single particle mass spectrometry)

IT Air pollution

(particulate, aerosol, anal. of; real-time detection method and system for identifying individual aerosol particles using bipolar single particle mass spectrometry)

IT Alarm devices

Biological warfare agents

Chemical warfare agents

Sampling

Time-of-flight mass spectrometry

(real-time detection method and system for identifying individual aerosol particles using bipolar single particle mass spectrometry)

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L136 ANSWER 18 OF 47 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2001:429352 CAPLUS

DOCUMENT NUMBER: 135:57096

TITLE: Effects of formaldehyde on the frog's mucociliary epithelium as a surrogate to evaluate air pollution effects on the respiratory epithelium

AUTHOR(S): Flo-Neyret, C.; Lorenzi-Filho, G.; Macchione, M.; Garcia, M. L. B.; Saldiva, P. H. N.

CORPORATE SOURCE: Departamento de Patologia Faculdade de Medicina, Universidade Federal de Sao Paulo, Sao Paulo, 01246-903, Brazil

SOURCE: Brazilian Journal of Medical and Biological Research (2001), 34(5), 639-643

CODEN: BJMRDK; ISSN: 0100-879X

PUBLISHER: Associacao Brasileira de Divulgacao Cientifica

DOCUMENT TYPE: Journal

LANGUAGE: English

ED Entered STN: 14 Jun 2001

AB The increasing use of alc. as an alternative fuel to gasoline or diesel can increase emission of formaldehyde, an organic gas that is irritant to the mucous membranes. The respiratory system is the major target of air pollutants and its major defense mechanism depends on the continuous activity of the cilia and the resulting constant transportation of mucous secretion. The present study was designed to evaluate the effects of formaldehyde on the ciliated epithelium through a relative large dose range around the threshold limit value adopted by the Brazilian legislation, namely 1.6 ppm (1.25-5 ppm). For this purpose, the isolated frog palate preparation was used as the target of toxic injury. Four groups of frog palates were exposed to diluted Ringer solution (control, N = 8) and formaldehyde diluted in Ringer solution at 3 different concns. (1.25, 2.5, and 5.0 ppm, N = 10 for each group). Mucociliary clearance and ciliary beat frequency decreased significantly in contact with formaldehyde at the concns. of 2.5 and 5.0 ppm after 60 min of exposure (P<0.05). Thus, relatively low concns. of formaldehyde, which is even below the Brazilian threshold limit value, are sufficient to cause short-term mucociliary impairment.

CC 4-3 (Toxicology)

ST Section cross-reference(s): 59

IT formaldehyde Rana palate cilia air pollution respiratory epithelium

IT Air pollution

Cilia

Palate

Rana catesbeiana

(effects of formaldehyde on frog mucociliary epithelium as surrogate to evaluate air pollution effects on respiratory epithelium)

IT Respiratory tract

(epithelium; effects of formaldehyde on frog mucociliary epithelium as surrogate to evaluate air pollution effects on respiratory epithelium)

IT 50-00-0, Formaldehyde, biological studies

RL: ADV (Adverse effect, including toxicity); POL (Pollutant); BIOL (Biological study); OCCU (Occurrence)

(effects of formaldehyde on frog mucociliary epithelium as surrogate to evaluate air pollution effects on respiratory epithelium)

REFERENCE COUNT: 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L136 ANSWER 19 OF 47 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2001:431342 CAPLUS

DOCUMENT NUMBER: 135:176512

TITLE: The further development of rainbow trout primary epithelial cell cultures as a diagnostic tool in ecotoxicology risk assessment

AUTHOR(S): Dowling, K.; Mothersill, C.

CORPORATE SOURCE: Radiation Science Centre, Dublin Institute of Technology, Dublin, Ire.

SOURCE: Aquatic Toxicology (2001), 53(3-4), 279-289

CODEN: AQTODG; ISSN: 0166-445X

PUBLISHER: Elsevier Science B.V.

DOCUMENT TYPE: Journal

LANGUAGE: English

ED Entered STN: 14 Jun 2001

AB The use of short-term cytotoxicity assays for the initial screening of chems. not only aids in establishing priorities for the selection of chems. that should be tested in vivo, but also decreases the time in which potential toxicants can be valued. Rainbow trout primary skin epithelial cell cultures are one such assay. Rainbow trout primary skin cell cultures contain two cell types, keratinocytes and goblet mucus cells. Two aquatic pollutants, copper and prochloraz were screened using this cell system. The influence of media composition on the effects of the aquatic pollutants was also studied by testing the chems. in both serum-containing and serum-free medium and the morphol. changes that occurred within the cell cultures recorded. The concentration of copper that causes a reduction of 90%

in the residual of day 3 growth of the primary cell culture system was found to be approx. 10 fold more than that of prochloraz. Prochloraz was found to cause a greater reduction in growth area when added to the primary cell culture system in serum-free medium than in serum-containing medium. Copper, in contrast, was found to exert reduced toxicity when added to the test cultures in serum-free medium compared with addition in serum-containing medium.

Prochloraz was found to kill the epithelial cells by a process of necrosis. Copper, was found to kill the epithelial cells by both necrosis and apoptosis in a ratio of 2:1. It was also observed that as the dose of both chems. increased, the number of goblet cells contained in the cell cultures decreased. A PAS stain was carried out to determine if the goblet cells were exocytosing their contents onto the cell culture surface. It was found that as chemical exposure increased the number of cells expressing positivity for mucus also increased. The results of this study add further evidence to support that primary cell cultures are a very appropriate model for toxicity risk assessment.

CC 4-1 (Toxicology)

Section cross-reference(s): 61

IT Toxicity

(aquatic; ecotoxicity risk assessment in relation to rainbow trout primary epithelial cell cultures as a diagnostic tool in ecotoxicol. risk assessment)

IT Proteins, general, biological studies

RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

- (blood; ecotoxicity risk assessment in relation to rainbow trout primary epithelial cell **cultures** as a diagnostic tool in ecotoxicol. risk assessment)
- IT Animal tissue culture
- Apoptosis
 - Bioassay
 - Cell death
 - Cell morphology
 - Cytotoxicity
 - Diagnosis
 - Ecotoxicity
 - Environmental pollution
 - Exocytosis
 - Oncorhynchus mykiss
 - Risk assessment
 - Water pollution
 - Xenobiotics
- (ecotoxicity risk assessment in relation to rainbow trout primary epithelial cell **cultures** as a diagnostic tool in ecotoxicol. risk assessment)
- IT Skin
- (epithelium; ecotoxicity risk assessment in relation to rainbow trout primary epithelial cell **cultures** as a diagnostic tool in ecotoxicol. risk assessment)
- IT Skin
- (goblet cell; ecotoxicity risk assessment in relation to rainbow trout primary epithelial cell **cultures** as a diagnostic tool in ecotoxicol. risk assessment)
- IT 7447-39-4, Cupric chloride, biological studies
- RL: ADV (Adverse effect, including toxicity); BIOL (Biological study) (ecotoxicity risk assessment in relation to rainbow trout primary epithelial cell **cultures** as a diagnostic tool in ecotoxicol. risk assessment)
- IT 7440-50-8, Copper, biological studies 67747-09-5, Prochloraz
- RL: ADV (Adverse effect, including toxicity); POL (Pollutant); BIOL (Biological study); OCCU (Occurrence) (ecotoxicity risk assessment in relation to rainbow trout primary epithelial cell **cultures** as a diagnostic tool in ecotoxicol. risk assessment)
- REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L136 ANSWER 20 OF 47 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2000:8546 CAPLUS

DOCUMENT NUMBER: 132:218213

TITLE: Internalization of Carcinogenic Lead Chromate Particles by Cultured Normal Human Lung Epithelial Cells: Formation of Intracellular Lead-Inclusion Bodies and Induction of Apoptosis

AUTHOR(S): Singh, Jatinder; Pritchard, Daryl E.; Carlisle, Diane L.; Mclean, John A.; Montaser, Akbar; Orenstein, Jan M.; Patierno, Steven R.

CORPORATE SOURCE: Department of Pharmacology, George Washington University Medical Center, Washington, DC, 20037, USA

SOURCE: Toxicology and Applied Pharmacology (1999), 161(3), 240-248

CODEN: TXAPA9; ISSN: 0041-008X

PUBLISHER: Academic Press

DOCUMENT TYPE: Journal

LANGUAGE: English

- ED Entered STN: 05 Jan 2000
- AB Occupational exposure to certain particulate hexavalent chromium [Cr(VI)] compds., such as lead chromate, has been associated with lung cancer and respiratory tract toxicity. The authors have previously shown that apoptosis is a major mode of death in cultured rodent cells treated with soluble sodium chromate and particulate lead chromate. Here the authors report the cellular and mol. effects of lead chromate and sodium chromate in normal human lung small airway epithelial (HSAE) cells, which may be one of the targets for Cr(VI)-induced lung cancer and respiratory tract toxicity. Phagocytosed lead chromate particles and intracellular lead-inclusion bodies (LIB) were observed by transmission electron microscopy and confirmed by X-ray anal. HSAE cells exposed to lead chromate and sodium chromate underwent dose-dependent apoptosis. The cellular uptake and genomic interactions of both Cr and lead (Pb) were examined by inductively coupled plasma mass spectrometry (ICPMS) coupled with a novel, direct-injection high-efficiency nebulizer (DIHEN). Using this approach, the authors have quantitated a dose-dependent formation of Cr-DNA adducts and DNA-associated Pb in lead chromate-treated HSAE cells. The formation of LIB in normal human lung cells exposed to lead chromate indicates that ionic Pb is released from the particles and thus might contribute to the cell toxicity caused by lead chromate. Internalization and dissoln. of lead chromate particles and the interaction of ionic Cr and Pb with DNA, may be components of the mechanism of lead chromate carcinogenesis. Lead chromate-induced apoptosis may be a mechanism to eliminate cells with chromium- and/or lead-damaged DNA. (c) 1999 Academic Press.
- CC 4-6 (Toxicology)
Section cross-reference(s): 59
- IT DNA
RL: ADV (Adverse effect, including toxicity); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(adducts, with chromium; lead chromate particle internalization by **cultured** normal human lung epithelial cells and formation of intracellular lead-inclusion bodies and induction of apoptosis)
- IT DNA
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(damage; lead chromate particle internalization by **cultured** normal human lung epithelial cells and formation of intracellular lead-inclusion bodies and induction of apoptosis)
- IT Carcinogens
(environmental; lead chromate particle internalization by **cultured** normal human lung epithelial cells and formation of intracellular lead-inclusion bodies and induction of apoptosis)
- IT Lung
(epithelium, cells; lead chromate particle internalization by **cultured** normal human lung epithelial cells and formation of intracellular lead-inclusion bodies and induction of apoptosis)
- IT Respiratory tract
(epithelium; lead chromate particle internalization by **cultured** normal human lung epithelial cells and formation of intracellular lead-inclusion bodies and induction of apoptosis)
- IT Mass spectrometry
(inductively coupled plasma; lead chromate particle internalization by **cultured** normal human lung epithelial cells and formation of intracellular lead-inclusion bodies and induction of apoptosis)
- IT Air pollution
(industrial; lead chromate particle internalization by **cultured** normal human lung epithelial cells and formation of intracellular

lead-inclusion bodies and induction of apoptosis)

IT Airborne particles
Apoptosis
Cell morphology
Cell nucleus
Cytoplasm
Cytotoxicity
Genotoxicity
Inclusion bodies
Lung, neoplasm
Occupational health
Transmission electron microscopy
X-ray spectroscopy
(lead chromate particle internalization by **cultured** normal human lung epithelial cells and formation of intracellular lead-inclusion bodies and induction of apoptosis)

IT Toxicity
(pulmonary, **respiratory**; lead chromate particle internalization by **cultured** normal human lung **epithelial** cells and formation of intracellular lead-inclusion bodies and induction of apoptosis)

IT Lung
(toxicity, **respiratory**; lead chromate particle internalization by **cultured** normal human lung **epithelial** cells and formation of intracellular lead-inclusion bodies and induction of apoptosis)

IT **Respiratory** tract
(toxicity; lead chromate particle internalization by **cultured** normal human lung **epithelial** cells and formation of intracellular lead-inclusion bodies and induction of apoptosis)

IT 7775-11-3, Sodium chromate
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(lead chromate particle internalization by **cultured** normal human lung epithelial cells and formation of intracellular lead-inclusion bodies and induction of apoptosis)

IT 7440-47-3D, Chromium, adducts with DNA, biological studies
RL: ADV (Adverse effect, including toxicity); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(lead chromate particle internalization by **cultured** normal human lung epithelial cells and formation of intracellular lead-inclusion bodies and induction of apoptosis)

IT 7439-92-1, Lead, biological studies 7440-47-3, Chromium, biological studies 7758-97-6, Lead chromate 11104-59-9, Chromate 18540-29-9, biological studies
RL: ADV (Adverse effect, including toxicity); POL (Pollutant); BIOL (Biological study); OCCU (Occurrence)
(lead chromate particle internalization by **cultured** normal human lung epithelial cells and formation of intracellular lead-inclusion bodies and induction of apoptosis)

REFERENCE COUNT: 55 THERE ARE 55 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L136 ANSWER 21 OF 47 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 1997:315762 CAPLUS

DOCUMENT NUMBER: 126:326672

TITLE: The effect of bacterial toxins on levels of intracellular adenosine nucleotides and human ciliary beat frequency

AUTHOR(S): Kanthakumar, K.; Taylor, G. W.; Cundell, D. R.;

Searched by Barb O'Bryen, STIC 2-2518

CORPORATE SOURCE: Dowling, R. B.; Johnson, M.; Cole, P. J.; Wilson, R.
Host Defence Unit, Imperial Coll. Sci., Tech. and
Med., National Heart and Lung Inst., London, SW3 6LR,
UK

SOURCE: Pulmonary Pharmacology (1996), 9(4), 223-230
CODEN: PUPHEX; ISSN: 0952-0600

PUBLISHER: Academic

DOCUMENT TYPE: Journal

LANGUAGE: English

ED Entered STN: 17 May 1997

AB The authors investigated whether reduction in intracellular adenosine nucleotides might be a common mechanism of action of other bacterial toxins which slow **ciliary beat**. Two other *Pseudomonas aeruginosa* toxins, 1-hydroxyphenazine (1-HP) and rhamnolipid, and two *Haemophilus influenzae* fractions produced by gel filtration of broth cultures were tested. The effect on human nasal epithelium **ciliary beat** frequency (CBF), and intracellular cAMP and ATP were measured, and the effect of the pharmacol. agents, dibutyryl cAMP and salmeterol, on these changes was assessed. 1-HP, rhamnolipid and the two *H. influenzae* fractions slowed CBF before there was significant release of lactate dehydrogenase from the cells. The toxins also caused a fall in intracellular cAMP and ATP. Dibutyryl cAMP and salmeterol at the concns. used do not increase baseline CBF, but diminished the fall in CBF and intracellular adenosine nucleotides. The cAMP and ATP levels in these studies were combined with those previously obtained with pyocyanin. There was a good correlation between cAMP and ATP levels and CBF. Bacterial toxins which slow CBF may act by causing a fall in intracellular adenosine nucleotides, and agents which stimulate cAMP may prevent toxin-induced slowing of **ciliary beat**.

CC 4-5 (Toxicology)

ST bacterial toxin adenosine nucleotide **ciliary beat**;
nasal **ciliated epithelium** human bacterial toxin

IT *Haemophilus influenzae*
Toxicity
(bacterial toxins effects on levels of intracellular adenosine nucleotides and human **ciliary beat** frequency)

IT Toxins
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(bacterial; bacterial toxins effects on levels of intracellular adenosine nucleotides and human **ciliary beat** frequency)

IT Nose
(**epithelium, ciliated**; bacterial toxins effects on levels of intracellular adenosine nucleotides and human **ciliary beat** frequency)

IT Cilia
(nasal epithelium; bacterial toxins effects on levels of intracellular adenosine nucleotides and human **ciliary beat** frequency)

IT Glycolipids
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(rhamnose-containing; bacterial toxins effects on levels of intracellular adenosine nucleotides and human **ciliary beat** frequency)

IT 528-71-2, 1-Hydroxyphenazine
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(bacterial toxins effects on levels of intracellular adenosine nucleotides and human **ciliary beat** frequency)

IT 56-65-5, 5'-ATP, biological studies 58-61-7D, Adenosine, nucleotides,
biological studies 60-92-4, CAMP

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL
(Biological study); PROC (Process)
(bacterial toxins effects on levels of intracellular adenosine
nucleotides and human **ciliary beat** frequency)

REFERENCE COUNT: 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L136 ANSWER 22 OF 47 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 1996:762561 CAPLUS

DOCUMENT NUMBER: 126:43660

TITLE: In vitro reconstituted tissue as an alternative to
human respiratory tract

AUTHOR(S): Emura, M.; Ochiai, A.; Hirohashi, S.

CORPORATE SOURCE: Inst. Experimentelle Pathologie, Med. Hochschule,
Hannover, D-30625, Germany

SOURCE: Toxicology Letters (1996), 88(1-3), 81-84

CODEN: TOLED5; ISSN: 0378-4274

PUBLISHER: Elsevier

DOCUMENT TYPE: Journal

LANGUAGE: English

ED Entered STN: 01 Jan 1997

AB The data obtained from in vitro systems utilizing human cells and tissues
should form a basic part of the information necessary for risk assessment.
The most important thing for such systems is, therefore, to simulate the
structures and functions of cells and tissues in the native organ as
closely as possible. In designing in vitro systems, there may be two
approaches-one aiming at the growth of cells in a primarily
two-dimensional fashion, the other allowing cells to form in
vivo-mimicking three-dimensional architectures. In cultures in which the
airway epithelial cells are growing in a two-dimensional fashion, some
functional and structural characteristics can be developed to a
considerable extent. However, there are some that cannot be developed or
expressed under that condition but require a three-dimensional growth
pattern. In this paper the authors explore the capacity of early to
long-term passage airway epithelial cells (human and hamster) to resume
architectures and functions existing in the native tissue in the specific
environments given in vitro.

CC 4-1 (Toxicology)

IT Lung
(**epithelial stem cell**; in vitro reconstituted tissue as an
alternative to human **respiratory tract**)

IT Bronchi
Lung
Respiratory tract
(**epithelium**; in vitro reconstituted tissue as an alternative
to human **respiratory tract**)

IT **Air pollution**
Cell junction
Endoplasmic reticulum
Environmental pollution
Golgi apparatus
Respiratory tract
(in vitro reconstituted tissue as an alternative to human respiratory
tract)

IT **Bioassay**
(in vitro tissue system; in vitro reconstituted tissue as an
alternative to human respiratory tract)

L136 ANSWER 23 OF 47 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 1995:520177 CAPLUS

DOCUMENT NUMBER: 122:258315
 TITLE: In vitro exposure of rabbit tracheal epithelium to SO₂: Effects on morphology and **ciliary beating**
 AUTHOR(S): Blanquart, C.; Giuliani, I.; Houcine, O.; Jeulin, C.; Guennou, C.; Marano, F.
 CORPORATE SOURCE: Lab. Cytophysiologie Toxicologie Cellulaire, Universite Paris VII Denis Diderot, Paris, 75251, Fr.
 SOURCE: Toxicology in Vitro (1995), 9(2), 123-32
 CODEN: TIVIEQ; ISSN: 0887-2333
 PUBLISHER: Elsevier
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 ED Entered STN: 02 May 1995
 AB The aim of this in vitro study was to characterize the direct effects of short-term exposure to low concns. of sulfur dioxide (SO₂) on both the morphol. and the physiol. of rabbit tracheal primary cultures. SEM studies revealed that ciliated cells exposed for 1 h to 10 ppm or 30 ppm SO₂ exhibited aggregated cilia. TEM revealed numerous swollen mitochondria in cells exposed to 30 ppm SO₂ for 1 h. This morphol. damage to cells was coupled with physiol. alterations. A 25% decrease in **ciliary beat** frequency (CBF) was measured in cells exposed to 30 ppm SO₂. This inhibition was partially reversible within 24 h. This SO₂ concentration also induced a significant depletion of cellular ATP content which was completely restored after a 24-h recovery period. A correlation was found between cellular ATP level depletion and CBF decrease.
 CC 4-3 (Toxicology)
 Section cross-reference(s): 59
 ST tracheal epithelium sulfur dioxide morphol; **ciliary beating** tracheal epithelium sulfur dioxide
 IT Air pollution
 Cell morphology
 (sulfur trioxide effect on trachea epithelium with respect to **ciliary beating** and morphol.)
 IT Trachea (anatomical)
 (epithelium, sulfur trioxide effect on trachea epithelium with respect to **ciliary beating** and morphol.)
 IT Trachea (anatomical)
 (epithelium, ciliated cell, sulfur trioxide effect on trachea epithelium with respect to **ciliary beating** and morphol.)
 IT 7446-09-5, Sulfur dioxide, biological studies
 RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
 (sulfur trioxide effect on trachea epithelium with respect to **ciliary beating** and morphol.)
 IT 7704-34-9, Sulfur, biological studies 14265-45-3, Sulfite 15181-46-1, Sulfite (HSO₃1-)
 RL: ADV (Adverse effect, including toxicity); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
 (sulfur trioxide effect on trachea epithelium with respect to **ciliary beating** and morphol.)
 IT 56-65-5, 5'-ATP, biological studies
 RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
 (sulfur trioxide effect on trachea epithelium with respect to **ciliary beating** and morphol.)

L136 ANSWER 24 OF 47 CAPLUS COPYRIGHT 2005 ACS on STN

Searched by Barb O'Bryen, STIC 2-2518

DOCUMENT NUMBER: 115:2801
TITLE: Assessing the toxicity of environmental gas pollutants
on **respiratory ciliated
epithelia**
CORPORATE SOURCE: Universite de Paris V, Lab. Diffus. Inelastique
Lumiere, Paris, Fr.
SOURCE: Report (1990), Order No. PB91-110841, 110 pp. Avail.:
NTIS
From: Gov. Rep. Announcé. Index (U. S.) 1991, 91(3),
Abstr. No. 106,921
DOCUMENT TYPE: Report
LANGUAGE: French
ED Entered STN: 12 Jul 1991
AB The authors developed a relatively easy and reproducible technique for
growing mammalian tracheal epithelial cultures, which they used to determine
criteria for assessing the action or toxic mols. on epithelial cells,
particularly ciliated cells. They designed a series of tests, including
cilio-inhibition, viability, and proliferation-inhibition tests, for
classifying the mols. tested. They also developed methods for measuring
cell physiol. activity, including measurement of: (1) **ciliary
beat frequency** through image anal. and phys. measurements; (2)
proliferation through image anal. and fluorescent marking; and (3)
viability through intracellular penetration of stains. This report
discusses the methods developed, which can be used for any kind of mol.,
as well as the specific study of three reference mols. known for their toxicity
to mammalian respiratory systems: acrolein, parathion, and caryololysine.
CC 4-3 (Toxicology)
ST Section cross-reference(s): 59
IT gas pollutant **respiratory tract epithelium**
IT **Air pollution**
(gas pollutants toxicity to **respiratory ciliated
epithelium** in relation to)
IT **Respiratory tract**
(**epithelium, ciliated, gas pollutants toxicity to**)
L136 ANSWER 26 OF 47 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1980:544183 CAPLUS
DOCUMENT NUMBER: 93:144183
TITLE: Response of **ciliated epithelium** to
ozone and sulfuric acid
AUTHOR(S): Grose, Elaine C.; Gardner, Donald E.; Miller,
Frederick J.
CORPORATE SOURCE: Health Effects Res., Northrop Serv., Inc., Research
Triangle Park, NC, 27709, USA
SOURCE: Environmental Research (1980), 22(2), 377-85
CODEN: ENVRAL; ISSN: 0013-9351
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 12 May 1984
AB The effects of O₃, H₂SO₄, and their interaction on ciliary activity were
investigated. Following in vivo exposure to various concns. of O₃ and
H₂SO₄, ciliary activity of isolated tracheal ring cultures was
microscopically determined under stroboscopic illumination. Assay of tracheal
rings immediately after a 2-h exposure to 880 µg H₂SO₄/m³ showed a
decrease in **ciliary beating frequency** from controls.
Following 72 h in vitro maintenance, there was still a depression in
ciliary activity of treatment cultures. In vivo recovery studies
indicated that ciliary activity had returned to the normal range 72 h
after exposure. Exposure to 196 µg O₃/m³ for 3 h resulted in no
significant difference from controls in ciliary activity. Expts. designed

ACCESSION NUMBER: 1992:77938 CAPLUS
DOCUMENT NUMBER: 116:77938
TITLE: Primary cultures of tracheal **epithelial**
cells for the evaluation of **respiratory**
toxicity
AUTHOR(S): Blanquart, C.; Romet, S.; Baeza, A.; Guennou, C.;
Marano, F.
CORPORATE SOURCE: Lab. Cytophysiol. Toxicol. Cell., Univ. Paris 7,
Paris, 75005, Fr.
SOURCE: Toxicology in Vitro (1991), 5(5-6), 499-502
CODEN: TIVIEQ; ISSN: 0887-2333
DOCUMENT TYPE: Journal
LANGUAGE: English

ED Entered STN: 06 Mar 1992

AB Cytotoxic assays have been carried out to evaluate toxic effects of
acrolein, mechlorethamine, parathion and paraoxon, using primary cultures
of rabbit tracheal epithelium obtained by the explant outgrowth technique.
The effect of drug exposure was first evaluated by the tetrazolium salt
assay, neutral red uptake and release of cellular lactate dehydrogenase.
These assays, previously developed for cell line cultures, were adapted to
this model. In addition, the authors present an easy and rapid method to
evaluate the effect of chems. on cell proliferation. This was performed
using an image anal. method, and was the most sensitive marker to determine the
acute toxicity of chems.

CC 4-1 (Toxicology)

Section cross-reference(s): 1

ST trachea **epithelium respiratory** toxicity evaluation;
acrolein **respiratory** toxicity evaluation trachea
epithelium; mechlorathamine **respiratory** toxicity
evaluation trachea **epithelium**; parathion **respiratory**
toxicity evaluation trachea **epithelium**; paraoxon
respiratory toxicity evaluation trachea **epithelium**

IT **Respiratory** tract

(chemical toxicity to, evaluation of, by trachea **epithelium**
toxicity assays)

IT Neoplasm inhibitors

(mechlorethamin as, toxicity of, to trachea **epithelium**, in
respiratory toxicity evaluation)

IT **Toxicity**

(of chems., to trachea **epithelium**, in **respiratory**
toxicity evaluation)

IT **Bioassay**

(of **respiratory** toxicity, in trachea **epithelium**)

IT Cell proliferation

(toxic chemical effect on, by trachea **epithelium**, in
respiratory toxicity evaluation)

IT Cytotoxic agents

(toxicity of, to trachea **epithelium**, in **respiratory**
toxicity evaluation)

IT Trachea (anatomical)

(**epithelium**, chemical toxicity to, in **respiratory**
toxicity evaluation)

IT 51-75-2 56-38-2, Parathion 107-02-8, Acrolein, biological studies
311-45-5

RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(toxicity of, to trachea **epithelium**, in **respiratory**
toxicity evaluation)

L136 ANSWER 25 OF 47 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1991:402801 CAPLUS

Searched by Barb O'Bryen, STIC 2-2518

to investigate the effects of a sequential exposure to O₃ followed by H₂SO₄ indicated a decrease in the **ciliary beating** frequency of exposed animals which was less than that observed with H₂SO₄ alone. Thus, combined action expts. are extremely relevant in assessing the toxicity of environmental pollutants.

CC 4-3 (Toxicology)

IT **Air pollution**
(by ozone and sulfuric acid, ciliary activity in relation to)

L136 ANSWER 27 OF 47 EMBASE COPYRIGHT (c) 2005 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2005234517 EMBASE
TITLE: Hyperoxia-induced changes in human airway epithelial cells:
The protective effect of perflubron.

AUTHOR: Babu P.B.R.; Chidekel A.; Shaffer T.H.
CORPORATE SOURCE: Dr. A. Chidekel, Department of Pediatrics, Alfred I. duPont
Hospital for Children, 1600 Rockland Road, Wilmington, DE
19803, United States. achidek@nemours.org

SOURCE: Pediatric Critical Care Medicine, (2005) Vol. 6, No. 2, pp.
188-194.

Refs: 53

ISSN: 1529-7535

COUNTRY: United States

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 015 Chest Diseases, Thoracic Surgery and Tuberculosis

029 Clinical Biochemistry

030 Pharmacology

037 Drug Literature Index

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 20050616

Last Updated on STN: 20050616

ABSTRACT: Objective: To determine the protective effect of perflubron (PFB), a type of perfluorochemical liquid, in hyperoxia-induced cellular injury in the human airway epithelial cells. Design: A controlled, in vitro laboratory study. Setting: Tertiary-care children's hospital. Subjects: Human airway epithelial cells. Interventions: Human airway epithelial cells, Calu-3 cells, grown on polycarbonate porous filters at an air-liquid interface culture were exposed to normoxic (FiCO(2) = 5%, balance air) or hyperoxic (FIO(2) = 95%, balance CO(2)) conditions. Hyperoxia-induced cellular changes were monitored by measuring transepithelial resistance (TER) of monolayers, histology of cells, total protein, and interleukin-8 (IL-8) secretion in apical surface fluid (ASF) washings. Under hyperoxic conditions, the protective effect of PFB was assessed by directly adding PFB liquid to the apical surface of monolayers. Measurements and main results: During hyperoxic gas-liquid interface culture, Calu-3 monolayers exhibited a loss of cellular integrity morphologically, decreased protein concentration, and IL-8 level in ASF washings. During hyperoxic PFB-liquid interface culture, there was an overall increase in TER value of monolayers, improved histology, decreased total protein secretion in ASF washings, and unaltered IL-8 secretion. Cytomorphologic observations of PFB-treated Calu-3 cells indicated the presence of varying numbers of differently sized intracellular vacuoles during both normoxic and hyperoxic conditions. Conclusions: We conclude that the air-liquid interface culture of Calu-3 may be helpful in understanding mechanisms of lung injuries caused in clinical practice, and PFB protects against hyperoxia-induced airway epithelial cell injury by promoting cellular integrity as well as cytologic modifications. PFB-liquid interface culture of Calu-3 may be a useful in vitro model for

studying the cytoprotective role of liquid ventilation. Copyright .COPYRGT.
2005 by the Society of Critical Care Medicine and the World Federation of
Pediatric Intensive and Critical Care Societies.

CONTROLLED TERM: Medical Descriptors:
*hyperoxia
*cell damage
*respiratory epithelium
epithelium cell
in vitro study
laboratory test
pediatric hospital
cell growth
filter
air
liquid
cell culture
monolayer culture
histology
cytokine release
cell surface
gas
cell structure
protein secretion
cell count
cell vacuole
lung injury
cell protection
liquid ventilation
human
controlled study
human cell
article
priority journal
Drug Descriptors:
*perfluorooctyl bromide: PD, pharmacology
polycarbonate
interleukin 8: EC, endogenous compound
(perfluorooctyl bromide) 423-55-2; (polycarbonate)
24936-68-3, 25766-59-0; (interleukin 8) 114308-91-7

CAS REGISTRY NO.:

L136 ANSWER 28 OF 47 EMBASE COPYRIGHT (c) 2005 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2004464305 EMBASE
TITLE: Regulation of human airway ciliary beat frequency by intracellular pH.
AUTHOR: Sutto Z.; Conner G.E.; Salathe M.
CORPORATE SOURCE: M. Salathe, Div. of Pulmonary/Critical Care Med., Univ. of Miami School of Medicine, 1600 NW 10th Avenue, Miami, FL 33136, United States. msalathe@miami.edu
SOURCE: Journal of Physiology, (15 Oct 2004) Vol. 560, No. 2, pp. 519-532.
Refs: 68
ISSN: 0022-3751 CODEN: JPHYA7
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 002 Physiology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20041119

Searched by Barb O'Bryen, STIC 2-2518

Last Updated on STN: 20041119

ABSTRACT: pH(i) affects a number of cellular functions, but the influence of pH(i) on mammalian ciliary beat frequency (CBF) is not known. CBF and pH(i) of single human tracheobronchial epithelial cells in submerged culture were measured simultaneously using video microscopy (for CBF) and epifluorescence microscopy with the pH-sensitive dye BCECF. Baseline CBF and pH(i) values in bicarbonate-free medium were 7.2 ± 0.2 Hz and 7.49 ± 0.02 , respectively (n = 63). Alkalization by ammonium pre-pulse to pH(i) 7.78 ± 0.02 resulted in a 2.2 ± 0.1 Hz CBF increase ($P < 0.05$). Following removal of NH_4Cl , pH(i) decreased to 7.24 ± 0.02 and CBF to 5.8 ± 0.1 Hz ($P < 0.05$). Removal of extracellular CO_2 to change pH(i) resulted in similar CBF changes. Pre-activation of cAMP-dependent protein kinase (10 μM forskolin), broad inhibition of protein kinases (100 μM H-7), inhibition of PKA (10 μM H-89), nor inhibition of phosphatases (10 μM cyclosporin + 1.5 μM okadaic acid) changed pH(i)-mediated changes in CBF, nor were they due to $[\text{Ca}^{2+}](i)$ changes. CBF of basolaterally permeabilized human tracheobronchial cells, re-differentiated at the air-liquid interface, was 3.9 ± 0.3 , 5.7 ± 0.4 , 7.0 ± 0.3 and 7.3 ± 0.3 Hz at basolateral i.e., intracellular pH of 6.8, 7.2, 7.6 and 8.0, respectively (n = 18). Thus, intracellular alkalization stimulates, while intracellular acidification attenuates human airway CBF. Since phosphorylation and $[\text{Ca}^{2+}](i)$ changes did not seem to mediate pH(i)-induced CBF changes, pH(i) may directly act on the ciliary motile machinery. .COPYRG. The Physiological Society 2004.

CONTROLLED TERM: Medical Descriptors:
 *respiratory epithelium
 *ciliary motility
 *cell pH
 cell function
 mammal
 tracheobronchial tree
 epithelium cell
 cell culture
 microscopy
 epifluorescence microscopy
 culture medium
 alkalinization
 statistical analysis
 enzyme inhibition
 cell permeabilization
 cell differentiation
 air
 liquid
 surface property
 basolateral membrane
 acidification
 human
 controlled study
 human tissue
 human cell
 article
 priority journal
 Drug Descriptors:
 dye
 bicarbonate
 ammonia
 carbon dioxide
 cyclic AMP
 protein kinase: EC, endogenous compound
 forskolin

Searched by Barb O'Bryen, STIC 2-2518.

phosphatase: EC, endogenous compound
 cyclosporin
 okadaic acid
 CAS REGISTRY NO.: (bicarbonate) 144-55-8, 71-52-3; (ammonia) 14798-03-9,
 51847-23-5, 7664-41-7; (carbon dioxide) 124-38-9,
 58561-67-4; (cyclic AMP) 60-92-4; (protein kinase)
 9026-43-1; (forskolin) 66575-29-9; (phosphatase) 9013-05-2;
 (cyclosporin) 79217-60-0; (okadaic acid) 78111-17-8

L136 ANSWER 29 OF 47 EMBASE COPYRIGHT (c) 2005 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2004381036 EMBASE
 TITLE: Exposure of differentiated airway epithelial cells to volatile smoke in vitro.
 AUTHOR: Beisswenger C.; Platz J.; Seifart C.; Vogelmeier C.; Bals R.
 CORPORATE SOURCE: Dr. R. Bals, Department of Internal Medicine, Division of Pulmonology, Hosp. of the University of Marburg, Baldingerstrasse 1, DE-35043 Marburg, Germany.
 SOURCE: bals@mail.uni-marburg.de
 Respiration, (2004) Vol. 71, No. 4, pp. 402-409.
 Refs: 27
 ISSN: 0025-7931 CODEN: RESPBD
 Switzerland
 COUNTRY: Journal; Article
 DOCUMENT TYPE: 005 General Pathology and Pathological Anatomy
 FILE SEGMENT: 015 Chest Diseases, Thoracic Surgery and Tuberculosis
 LANGUAGE: English
 SUMMARY LANGUAGE: English
 ENTRY DATE: Entered STN: 20040924
 Last Updated on STN: 20040924

ABSTRACT: Background: Cigarette smoke (CS) is the predominant pathogenetic factor in the development of chronic bronchitis and chronic obstructive pulmonary disease. The knowledge about the cellular and molecular mechanisms underlying the smoke-induced inflammation in epithelial cells is limited. Objectives: The aim of this study was to develop an in vitro model to monitor the effects of volatile CS on differentiated airway epithelial cells. Methods: The airway epithelial cell line MM-39 and primary human bronchial epithelial cells were cultivated as air-liquid interface cultures and exposed directly to volatile CS. We used two types of exposure models, one using ambient air, the other using humidified and warm air. Cytokine levels were measured by quantitative PCR and ELISA. Phosphorylation of p38 MAP kinase was assessed by Western blot analysis. To reduce the smoke-induced inflammation, antisense oligonucleotides directed against the p65 subunit of NF- κ B were applied. Results: Exposure of epithelia to cold and dry air resulted in a significant inflammatory response. In contrast, exposure to humidified warm air did not elicit a cellular response. Stimulation with CS resulted in upregulation of mRNA for IL-6 and IL-8 and protein release. Exposure to CS combined with heat-inactivated bacteria synergistically increased levels of the cytokines. Reactions of differentiated epithelial cells to smoke are mediated by the MAP kinase p38 and the transcription factor NF- κ B. Conclusions: We developed an exposure model to examine the consequences of direct exposure of differentiated airway epithelial cells to volatile CS. The model enables to measure the cellular reactions to smoke exposure and to determine the outcome of therapeutic interventions. Copyright .COPYRG. 2004 S. Karger AG, Basel.

CONTROLLED TERM: Medical Descriptors:
 *smoke
 *chronic obstructive lung disease: ET, etiology
 respiratory epithelium

Searched by Barb O'Bryen, STIC 2-2518

cell differentiation
disease model
cell culture
ambient air
humidity
enzyme linked immunosorbent assay
quantitative analysis
polymerase chain reaction
inflammation
enzyme phosphorylation
upregulation
protein secretion
human
controlled study
human cell
article
priority journal
Drug Descriptors:
*volatile agent
cytokine: EC, endogenous compound
mitogen activated protein kinase: EC, endogenous compound
immunoglobulin enhancer binding protein: EC, endogenous compound
interleukin 6: EC, endogenous compound
interleukin 8: EC, endogenous compound
(mitogen activated protein kinase) 142243-02-5;
(interleukin 8) 114308-91-7

CAS REGISTRY NO.:

L136 ANSWER 30 OF 47 EMBASE COPYRIGHT (c) 2005 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2002341737 EMBASE

TITLE: Effects of hexamethylene diisocyanate exposure on human airway epithelial cells: In vitro cellular and molecular studies.

AUTHOR: Wisniewski A.V.; Liu Q.; Miller J.-J.; Magoski N.; Redlich C.A.

CORPORATE SOURCE: A.V. Wisniewski, Yale University School of Medicine, Department of Internal Medicine, 333 Cedar Street, New Haven, CT 06520, United States. adam.wisniewski@yale.edu

SOURCE: Environmental Health Perspectives, (2002) Vol. 110, No. 9, pp. 901-907.

Refs: 35

ISSN: 0091-6765 CODEN: EVHPAZ

COUNTRY: United States

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 015 Chest Diseases, Thoracic Surgery and Tuberculosis
029 Clinical Biochemistry
046 Environmental Health and Pollution Control
052 Toxicology

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 20021017

Last Updated on STN: 20021017

ABSTRACT: In this study we developed an in vitro exposure model to investigate the effects of hexamethylene diisocyanate (HDI) on human airway epithelial cells at the cellular and molecular level. We used immunofluorescence analysis (IFA) to visualize the binding and uptake of HDI by airway epithelial cell lines (A549 and NCI-NCI-H292) and microarray technology to identify HDI sensitive genes. By IFA, we observed that subcytotoxic concentrations of HDI form microscopic micelles that appear to be taken up by cells over a 3-hr

Searched by Barb O'Bryen, STIC 2-2518

period postexposure. Microarray analysis (4.6K genes) of parallel cultures identified four genes (thioredoxin reductase, dihydrodiol dehydrogenase, TG interacting factor, and stanniocalcin) whose mRNA levels were up-regulated after HDI exposure. Northern analysis was used to confirm that HDI increased message levels of these four genes and to further explore the dose dependence and kinetics of the response. The finding that HDI exposure increases thioredoxin reductase expression supports previous studies suggesting that HDI alters thiol-redox homeostasis, an important sensor of cellular stress. Another of the HDI-increased genes, a dihydrodiol dehydrogenase, encodes a protein previously shown to be specifically susceptible to HDI conjugation, and known to detoxify other hydrocarbons. Together, the data describe a novel approach for investigating the effects of HDI binding and uptake by human airway epithelial cells and begin to identify genes that may be involved in the acute response to exposure.

CONTROLLED TERM: Medical Descriptors:
 environmental exposure
 respiratory epithelium
 epithelium cell
 in vitro study
 immunofluorescence test
 binding kinetics
 transport kinetics
 cell line
 DNA microarray
 gene identification
 cytotoxicity
 concentration (parameters)
 microscopy
 micelle
 cell culture
 Northern blotting
 gene expression
 oxidation reduction state
 stress
 human
 controlled study
 human cell
 article
 nucleotide sequence
 priority journal
 Drug Descriptors:
 *hexamethylene diisocyanate: TO, drug toxicity
 DNA: EC, endogenous compound
 thioredoxin reductase: EC, endogenous compound
 dihydrodiol dehydrogenase: EC, endogenous compound
 hypocalcin: EC, endogenous compound
 messenger RNA: EC, endogenous compound
 (hexamethylene diisocyanate) 11142-52-2, 822-06-0; (DNA)
 9007-49-2; (thioredoxin reductase) 9074-14-0; (dihydrodiol
 dehydrogenase) 37255-32-6; (hypocalcin) 76687-96-2
 GENBANK AA085318 referred number; GENBANK AA453335 referred
 number; GENBANK R83270 referred number; GENBANK R93124
 referred number

CAS REGISTRY NO.:

GENE NUMBER:

L136 ANSWER 31 OF 47 EMBASE COPYRIGHT (c) 2005 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2001001548 EMBASE

TITLE: Coal fly ash and mineral dust for toxicology and particle characterization studies: Equipment and methods for PM2.5-

and PM1-enriched samples.
AUTHOR: Veranth J.M.; Smith K.R.; Aust A.E.; Dansie S.L.; Griffin J.B.; Hu A.A.; Huggins M.L.; Lighty J.S.
CORPORATE SOURCE: J.M. Veranth, Dept. of Chemical/Fuels Engineering, University of Utah, 207 KRC 1495 East 100 South, Salt Lake City, UT 84112-1114, United States
SOURCE: Aerosol Science and Technology, (2000) Vol. 32, No. 2, pp. 127-141.
Refs: 39
ISSN: 0278-6826 CODEN: ASTYDQ
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 046 Environmental Health and Pollution Control
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20010111
Last Updated on STN: 20010111
ABSTRACT: Laboratory methods to produce particle samples from known, reproducible sources with sufficient mass to perform both detailed characterization and replicated in vitro toxicological assays are described. These samples are being used to study the ability of inhalable particles to produce abnormal concentrations of intracellular iron, resulting in the production of reactive oxygen species in cultured airway epithelial cells. Bulk samples of size fractionated particles from laboratory-generated coal fly ash and from simulated fugitive mining tailings and road dust were collected as surrogates for important sources of iron-bearing particles in the ambient air. An Andersen cascade impactor was used to produce particle samples enriched in three size ranges: >10 μm , 10-2.5 μm , and <2.5 μm aerodynamic diameter. A multijet preseparator and rectangular slot virtual impactor were used to produce a fraction enriched in particles below 1 μm . Data on the particle production conditions, production rates, and particle sample quality are provided to illustrate the feasibility of the experimental approach. The amount of iron mobilized from particles by a physiologically-relevant chelator does not correlate with the total iron. This supports the hypothesis that particle characteristics and iron speciation are important for the production of abnormal iron concentrations in cultured type A549 human airway epithelial cells. Comparison of results obtained with these surrogate particles to previous work with urban particulate standard reference materials (SRM 1648 and SRM 1649) suggests particle sources and size fractions that should be emphasized for detailed characterization of particle morphology and mineralogy.

CONTROLLED TERM: Medical Descriptors:
*fly ash
*mineral dust
*toxicology
particle size
bioaccumulation
cell culture
aerosol
in vitro study
inhalation
respiratory epithelium
simulation
mining
ambient air
feasibility study
hypothesis
human
controlled study
human cell

article
priority journal
Drug Descriptors:
*coal
*mineral
iron
iron chelate

CAS REGISTRY NO.: (iron) 14093-02-8, 53858-86-9, 7439-89-6

L136 ANSWER 32 OF 47 PASCAL COPYRIGHT 2005 INIST-CNRS. ALL RIGHTS RESERVED.
on STN DUPLICATE 8

ACCESSION NUMBER: 1995-0499318 PASCAL

COPYRIGHT NOTICE: Copyright .COPYRG. 1995 INIST-CNRS. All rights reserved.

TITLE (IN ENGLISH): Tracheal epithelial cells in vitro as a model to study genotoxicity of **airborne** particulates

AUTHOR: HORNBERG C.; SEEMAYER N. H.
REINHARDT Christoph A. (ed.); BENFORD Diane J. (ed.);
BLAAUBOER Bas J. (ed.)

CORPORATE SOURCE: Medical inst. environmental hyg., Heinrich-Heine-Univ.
40223 Duesseldorf, Germany, Federal Republic of
Swiss inst. alternatives animal testing, 8005 Zuerich,
Switzerland

SOURCE: Toxicology in vitro, (1995), 9(4), 397-402 [5 p.], 30
refs.
Conference: 8 INVITOX 94 : International wokshop on in
vitro toxicology, Kartause Ittingen (Switzerland), 20
Sep 1994

ISSN: 0887-2333 CODEN: TIVIEQ

Journal; Conference

DOCUMENT TYPE:

BIBLIOGRAPHIC LEVEL: Analytic

COUNTRY: United Kingdom

LANGUAGE: English

AVAILABILITY: INIST-21203, 354000054146370070

ABSTRACT: The major target site of **airborne** particulates is the tracheobronchial **epithelium** of the **respiratory** tract. It is also the origin of the most common cancer in man, bronchogenic carcinoma. Rodent tracheal epithelial cells in **culture** can be used to study the genotoxic activity of **airborne** particulates leading to mutation and cancer. **Airborne** particulates were collected in the heavily industrialized Rhine-Ruhr region using a high volume sampler HVS 150 (Stroehlein Instruments) equipped with glass fibre filters. Chemical substances were extracted with di-chloromethane or methanol and quantitatively transferred to dimethyl sulfoxide for tissue **culture** experiments. Tracheal epithelial cells of the Syrian golden hamster and the Wistar rat were dissociated by pronase treatment and cultivated in a 'complex' medium. The induction of sister chromatid exchanges was used as a sensitive **bioassay** for detection of genotoxic activity of **airborne** particulates. Extracts of **airborne** particulates led to a dose-related highly significant induction of sister chromatid exchanges in cell **cultures** of tracheal epithelial cells of the hamster and the rat. Even quantities of chemical substances equivalent to

CLASSIFICATION CODE:
CONTROLLED TERM:

airborne particulates from less than 1 m.sup.3
air were markedly genotoxic.
002B03N; Life sciences; Medical sciences; Toxicology
Suspended particle; Toxicity; Carcinogen;
Carcinogenicity testing; Investigation method;
Epithelial cell; Trachea; Dose activity relation; DNA;
Sister chromatid exchange; Chromosomal aberration;
Mutagenicity testing
Respiratory disease

BROADER TERM:

L136 ANSWER 33 OF 47 PASCAL COPYRIGHT 2005 INIST-CNRS. ALL RIGHTS RESERVED.
on STN

ACCESSION NUMBER:
COPYRIGHT NOTICE:

2000-0063051 PASCAL
Copyright .COPYRG. 2000 INIST-CNRS. All rights
reserved.

TITLE (IN ENGLISH):

Effects of aqueous extracts of PM.sub.1.sub.0 filters
from the Utah Valley on human airway epithelial cells
FRAMPTON M. W.; GHIO A. J.; SAMET J. M.; CARSON J. L.;
CARTER J. D.; DEVLIN R. B.

AUTHOR:

CORPORATE SOURCE:

Department of Medicine and Environmental Medicine,
University of Rochester School of Medicine and
Dentistry, Rochester, New York 14642, United States;
Human Studies Division, United States Environmental
Protection Agency, Chapel Hill, North Carolina 27599,
United States; Center for Environmental Medicine and
Lung Biology, University of North Carolina, Chapel
Hill, North Carolina 27599, United States
American journal of physiology. Lung cellular and
molecular physiology, (1999), 21(5), L960-L967, 31
refs.

SOURCE:

ISSN: 1040-0605

DOCUMENT TYPE:
BIBLIOGRAPHIC LEVEL:
COUNTRY:
LANGUAGE:
AVAILABILITY:
ABSTRACT:

Journal
Analytic
United States
English
INIST-22200, 354000080406110140
We hypothesized that the reduction in hospital
respiratory admissions in the Utah Valley during
closure of a local steel mill in 1986-1987 was
attributable in part to decreased toxicity of ambient
air particles. **Sampling** filters for
particulate matter < 10 µm (PM.sub.1.sub.0) were
obtained from a Utah Valley monitoring station for the
year before (year 1), during (year 2), and after (year
3) the steel mill closure. Aqueous extracts of the
filters were analyzed for metal content and oxidant
production and added to **cultures** of human
respiratory epithelial (BEAS-2B)
cells for 2 or 24 h. Year 2 dust contained the lowest
concentrations of soluble iron, copper, and zinc and
showed the least oxidant generation. Only dust from
year 3 caused cytotoxicity (by microscopy and lactate
dehydrogenase release) at 500 µg/ml. Year 1 and
year 3, but not year 2, dust induced expression of
interleukin-6 and -8 in a dose-response fashion. The
effects of ambient air particles on human respiratory
epithelial cells vary significantly with time and
metal concentrations.

CLASSIFICATION CODE:
CONTROLLED TERM:

002B03L05; Life sciences; Medical sciences; Toxicology
Heavy metal; Pollutant; Toxicity; Epithelial cell;

BROADER TERM: Respiratory tract; Interleukin 8; Interleukin 6;
Aqueous solution; Extract; Filter; Human; Utah
Air pollution; Respiratory system;
United States; North America; America

L136 ANSWER 34 OF 47 Elsevier BIOBASE COPYRIGHT 2005 Elsevier Science B.V.
on STN DUPLICATE

ACCESSION NUMBER: 1994080637 ESBIODASE

TITLE: In vitro exposure of tracheobronchial epithelial cells
and of tracheal explants to ozone

AUTHOR: Tarkington B.K.; Wu R.; Sun W.-M.; Nikula K.J.; Wilson
D.W.; Last J.A.

CORPORATE SOURCE: J.A. Last, Calif. Regional Primate Res. Center,
University of California, Davis, CA 95616-8542, United
States.

SOURCE: Toxicology, (1994), 88/1-3 (51-68)
CODEN: TXCYAC ISSN: 0300-483X

DOCUMENT TYPE: Journal; Article

COUNTRY: Ireland

LANGUAGE: English

SUMMARY LANGUAGE: English

ABSTRACT: An in vitro system for exposing **respiratory**
epithelial cells or explant tissues to ozone
has been developed and characterized. This system is
designed to generate and monitor consistent,
reproducible levels of ozone, over a range of
concentrations, in a humidified atmosphere, and to
allow an exposure time of 24 h or longer. Based on
chemical analysis, highly reproducible concentrations
of ozone are delivered throughout the chamber, with a
coefficient of variation of < 5% between five
replicate vials exposed to 0.5 ppm of ozone for 50
min. The viability of **cultured** human
tracheobronchial epithelial cells, as measured by the
ability to oxidize a vital dye, and of rat tracheal
epithelium, as measured by total numbers of necrotic
cells in tracheal explants, after ozone exposure was
examined in this system. Responses of **cultured**
cells to ozone exposure as measured by
bioassay were consistent with the observed low
level of variability of ozone concentration between
replicate incubation dishes or vials. Responses of
cultured cells to ozone were proportional to
duration of exposure and inversely proportional to the
volume of medium covering the cells. We conclude that
this newly developed in vitro exposure system will
allow relatively simple and convenient exposure of
cultured cells or organs to ozone or other
gaseous agents under highly controlled and
reproducible conditions.

CLASSIFICATION CODE: 90.10.3 TOXICOLOGY: METHODS IN CLINICAL AND
EXPERIMENTAL TOXICOLOGY: Toxicity Tests
90.5.1.2 TOXICOLOGY: EXPERIMENTAL TOXICOLOGY (by
agent): Industrial and **Environmental**
Pollutants: Other inorganic
90.6.6 TOXICOLOGY: CLINICAL AND EXPERIMENTAL
TOXICOLOGY (by target organ): Respiratory

SUPPLEMENTARY TERM: Ozone; Cell **culture**; **Air**
pollution

L136 ANSWER 35 OF 47 Elsevier BIOBASE COPYRIGHT 2005 Elsevier Science B.V.
on STN

ACCESSION NUMBER: 1999279528 ESBIOBASE
TITLE: Effects of aqueous extracts of PM.sub.1.sub.0 filters from the Utah Valley on human airway epithelial cells
AUTHOR: Frampton M.W.; Ghio A.J.; Samet J.M.; Carson J.L.; Carter J.D.; Devlin R.B.
CORPORATE SOURCE: M.W. Frampton, Pulmonary and Critical Care Unit, University of Rochester, School of Medicine/Dentistry, 601 Elmwood Ave., Rochester, NY 14642-8692, United States.
SOURCE: American Journal of Physiology - Lung Cellular and Molecular Physiology, (1999), 277/5 21-5 (L960-L967), 31 reference(s)
CODEN: APLPE7 ISSN: 1040-0605
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
ABSTRACT: We hypothesized that the reduction in hospital respiratory admissions in the Utah Valley during closure of a local steel mill in 1986-1957 was attributable in part to decreased toxicity of ambient air particles. **Sampling** filters for particulate matter < 10 µm (PM.sub.1.sub.0) were obtained from a Utah Valley monitoring station for the year before (year 1), during (year 2), and after (year 3) the steel mill closure. Aqueous extracts of the filters were analyzed for metal content and oxidant production and added to **cultures** of human **respiratory epithelial** (BEAS-2B) cells for 2 or 24 h. Year 2 dust contained the lowest concentrations of soluble iron, copper, and zinc and showed the least oxidant generation. Only dust from year 3 caused cytotoxicity (by microscopy and lactate dehydrogenase release) at 500 µg/ml. Year 1 and year 3, but not year 2, dust induced expression of interleukin-6 and -8 in a dose-response fashion. The effects of ambient air particles on human respiratory epithelial cells vary significantly with time and metal concentrations.
CLASSIFICATION CODE: 89.5.1.7 CELL AND DEVELOPMENTAL BIOLOGY: CELL TYPES AND BIOLOGY: Cell Types: Epithelial and endothelial cells
SUPPLEMENTARY TERM: **Air pollution**; Metals; Interleukin-6; Interleukin-8; Toxicity

L136 ANSWER 36 OF 47 Elsevier BIOBASE COPYRIGHT 2005 Elsevier Science B.V.
on STN

ACCESSION NUMBER: 1999251260 ESBIOBASE
TITLE: Intranasal toxicity of BMS-181885, a novel 5-HT1 agonist
AUTHOR: Schulze G.E.; Proctor J.E.; Dominick M.A.; Weiss A.E.; Flint O.P.; Srinivas N.R.; Durham S.K.; Schilling B.E.
CORPORATE SOURCE: Dr. G.E. Schulze, Bristol-Myers Squibb Co., 6000 Thompson Road, Syracuse, NY 13057, United States. E-mail: schulzeg@bms.com
SOURCE: International Journal of Toxicology, (1999), 18/5 (285-296), 48 reference(s)
CODEN: IJTOFN ISSN: 1091-5818

DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
ABSTRACT: One-month intranasal toxicity studies were conducted with BMS-181885 at doses of 1.5, 9, or 15 mg/animal/day in rats and 4, 24, or 40 mg/animal/day in monkeys. A 1-month intermittent intranasal toxicity study was also conducted in monkeys at doses of 3, 6, and 12 mg/animal 3 days per week. BMS- 181885 was generally well tolerated in rats but resulted in dose-dependent nasal mucosal injury, primarily characterized by subacute inflammation of the nasal mucosa, and degeneration, single-cell necrosis, and/or erosion of the olfactory epithelium and, to a lesser extent, the **respiratory epithelium**. In monkeys, daily BMS-181885 administration was well tolerated and produced similar dose-dependent nasal injury primarily characterized by subacute inflammation of the nasal mucosa with degeneration and erosion of the olfactory epithelium. In a separate experiment, intermittent administration also resulted in dose-dependent nasal injury. In **cultured** rat nasal mucosal cells, BMS-181885 was toxic to olfactory epithelial cells with a range of mean IC_{sub}50s between 44 and 291 μ M. In contrast, BMS-181885 had no effect on **respiratory epithelial** cells up to its maximum solubility. Cytochrome P450 inhibition had no effect on the toxicity of BMS-181885 in olfactory epithelial cells but produced dose-dependent toxicity in **respiratory epithelial** cells, which was not present previously. The in vitro data suggest that parent drug, rather than a toxic metabolite, caused the drug-associated nasal mucosal injury.

CLASSIFICATION CODE: 90.4.1.3 TOXICOLOGY: CLINICAL TOXICOLOGY (by agent): Industrial and **Environmental Pollutants**: Pesticides and agricultural chemicals
90.6.1 TOXICOLOGY: CLINICAL AND EXPERIMENTAL TOXICOLOGY (by target organ): Neuro-**sensory**
90.10.3 TOXICOLOGY: METHODS IN CLINICAL AND EXPERIMENTAL TOXICOLOGY: Toxicity Tests

SUPPLEMENTARY TERM: 5-HT agonist; Intranasal toxicity; In vitro; In vivo; Metabolism; Monkeys; Nasal toxicity; P450; Rats; Safety assessment; Serotonin

L136 ANSWER 37 OF 47 NIOSHTIC on STN
ACCESSION NUMBER: 1997:180032 NIOSHTIC
DOCUMENT NUMBER: NIOSH-00210743
TITLE: Toxic Effects of Sulfur Mustard on **Respiratory Epithelial** Cells in **Culture**
AUTHOR(S): Chevillard, M.; Laine, P.; Robineau, P.; Puchelle, E.
SOURCE: Cell Biology and Toxicology, Vol. 8, No. 2, pages 171-181, 24 references
CODEN: CBTOE2
PUBLICATION DATE: Apr 1992
DOCUMENT TYPE: Journal
LANGUAGE: ENGLISH
ABSTRACT:

The toxicity of sulfur-mustard (505-60-2) (SM) to respiratory epithelial cells was studied in-vitro. Respiratory epithelial cells isolated from male New-Zealand-white-rabbit tracheas were cultured and incubated with 0.1, 1, or 10 millimolar (mM) SM for 2 minutes. The effects on **ciliary ***beat***** frequency were determined 30, 60, or 90 minutes and 2, 3, 4, 5, or 6 hours after treatment using a videomicroscopic technique. The cultures were observed for morphological changes by optical and electron microscopy. Control cultures developed a layer of adherent epithelial cells that progressively increased to form an outgrowth of differentiated and undifferentiated cells. The 1 and 10mM SM exposures arrested **ciliary beating** within 30 and 10 minutes, respectively. The 0.1mM exposure caused an irreversible arrest of **ciliary beating** in the outgrowth zone after 90 minutes in two of six cultures. **Ciliary beating** frequency was not affected by 0.1mM SM in the other four cultures. Morphological changes were assessed only in cultures treated with 0.1mM SM because of the less drastic effect on **ciliary beating** frequency. SM induced vacuolization of the ciliated cells after 1 hour. The outgrowth zone became less adherent after 2 hours and tended to roll up after 4 hours. The outgrowth zone eventually became completely detached. Treatment related ultrastructural changes included mitochondrial swelling that appeared after 1 hour and cytoplasmic abnormalities such as increases in the number of intermediate filaments and the development of organelle free zones near the cell periphery that appeared at 6 hours. The authors conclude that SM profoundly affects the viability of **respiratory epithelial cell cultures**. The sudden arrest of **ciliary beating** more likely reflects the death of ciliated respiratory epithelial cells than a specific ciliotoxic effect of SM.

CONTROLLED TERM: Mustard gas; Mammalian cells; In vitro studies; Cytotoxic effects; Dose response; Cellular function; Ultrastructure; Histomorphology; **Chemical warfare agents**
CAS REGISTRY NO.: 505-60-2 (sulfur-mustard)

L136 ANSWER 38 OF 47 INSPEC (C) 2005 IEE on STN
ACCESSION NUMBER: 2000:6665594 INSPEC
DOCUMENT NUMBER: A2000-18-8760F-022; B2000-09-7510J-031
TITLE: Homodyne mixing of scattered light as a novel technique for the measurement of **ciliary beat** frequency.
AUTHOR: Wilson, M.J. (Dept. of Med. Phys., Univ. Hosp., Birmingham, UK); Drake-Lee, A.; Wang, R.
SOURCE: Proceedings of the SPIE - The International Society for Optical Engineering (2000) vol.3915, p.170-7. 20 refs..
Published by: SPIE-Int. Soc. Opt. Eng
Price: CCCC 0277-786X/2000/\$15.00
CODEN: PSISDG ISSN: 0277-786X
SICI: 0277-786X(2000)3915L:170:HMSL;1-Q
Conference: Coherence Domain Optical Methods in Biomedical Science and Clinical Applications IV. San Jose, CA, USA, 24-26 Jan 2000
Sponsor(s): SPIE; IBOS Int. Biomed. Opt. Soc
DOCUMENT TYPE: Conference Article; Journal
TREATMENT CODE: Experimental
COUNTRY: United States
LANGUAGE: English
ABSTRACT: Cilia are finger like organelles present throughout the **epithelium** of the human **respiratory** tract. Typically 5-6 μ m long,

with a density of around $8/\mu m^2$, they 'beat' at frequencies of 10-20 Hz and propel an overlying mucus layer. The mucus traps airborne particulates providing an essential respiratory cleaning/infection control mechanism. A novel method for measurement of ciliary beat frequency in-vivo is presented. Homodyne mixing of scattered coherent light promises a relatively large signal with minimal sensitivity to vibrational when compared with existing methods: Vibrational susceptibility is minimized since all scattered pathlengths are similarly affected, while phase discrepancies introduced by the muco-ciliary surface produce high contrast speckle giving good signal quality. The need to illuminate a relatively small area of epithelium to give large speckle has been addressed using a 'shortened' gradient index rod as a combined delivery/focusing device. Results are good when the ciliary surface reflectivity is increased. However the relatively low reflection coefficient of the unprepared surface allows homodyne mixing from deeply scattered light giving a speckle structure too small to resolve and consequent signal loss. Evidence is presented that demonstrates how a source with limited temporal coherence might be used to suppress the interference from these 'longer pathlength' photons.

CLASSIFICATION CODE:

A8760F Optical and laser radiation (medical uses);
 A8725D Biological transport; cellular and subcellular transmembrane physics; A4225F Optical diffraction and scattering; A8750B Interactions of biosystems with radiations; A8770E Patient diagnostic methods and instrumentation; A4225H Optical interference and speckle; A0760L Optical interferometry; A4225K Optical coherence; A4281P Fibre optic sensors; fibre gyros; B7510J Optical and laser radiation (biomedical imaging/measurement); B7230E Fibre optic sensors
 CELL MOTILITY; FIBRE OPTIC SENSORS; LASER APPLICATIONS IN MEDICINE; LIGHT COHERENCE; LIGHT SCATTERING; SPECKLE

CONTROLLED TERM:

SUPPLEMENTARY TERM:

finger like organelles; homodyne mixing; phase discrepancies; scattered coherent light; epithelium; human respiratory tract; overlying mucus layer; **airborne particulates**; essential respiratory cleaning/infection control mechanism; **ciliary beat frequency in-vivo**; vibrational susceptibility; scattered pathlength; muco-ciliary surface; signal quality; shortened gradient index rod; combined delivery/focusing device; ciliary surface reflectivity; reflection coefficient; unprepared surface; deeply scattered light; speckle structure; signal loss; limited temporal coherence; longer pathlength photons

L136 ANSWER 39 OF 47 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on
 STN DUPLICATE 2

ACCESSION NUMBER: 2001:311226 BIOSIS

DOCUMENT NUMBER: PREV200100311226

TITLE: Genotoxicity of nitroso compounds and sodium dichromate in a model combining organ cultures of human nasal epithelia and the comet assay.

Searched by Barb O'Bryen, STIC 2-2518

AUTHOR(S): Kleinsasser, Norbert H. [Reprint author]; Gamarra, Fernando; Bergner, Albrecht; Wallner, Barbara C.; Harreus, Ulrich A.; Juchhoff, Jutta; Kastenbauer, Ernst R.; Huber, Rudolf M.

CORPORATE SOURCE: Klinisch Experimentelle Onkologie, Klinik und Poliklinik fuer Hals-, Nasen- und Ohrenkranke, Ludwig-Maximilians-Universitaet Muenchen, Pettenkoferstrasse 4a, D-80336, Muenchen, Germany
norbert.kleinsasser@hno.med.uni-muenchen.de

SOURCE: ORL (Basel), (May-June, 2001) Vol. 63, No. 3, pp. 141-147. print.
CODEN: ORLJAH. ISSN: 0301-1569.

DOCUMENT TYPE: Article

LANGUAGE: English

ENTRY DATE: Entered STN: 27 Jun 2001
Last Updated on STN: 19 Feb 2002

ABSTRACT: Genotoxic effects of xenobiotics are a possible step in tumor initiation in the mucosa of the upper aerodigestive tract. Using the comet assay, detecting genotoxicity in human tissue has been restricted to single incubations in vitro, but in vivo most xenobiotics harm their target in a repetitive or chronic manner. Therefore, we propose a model, which provides repetitive incubations in human upper aerodigestive tract mucosa cultures. ***Samples*** of human inferior nasal turbinate mucosa (n=25) were cultured according to a modified version of a technique originally described by Steinsvag. On day 1 fresh samples and on days 7, 9 and 11 organ cultures were incubated with N-nitrosodiethylamine (NDEA), sodium dichromate (Na2Cr2O7) and N'-methyl-N-nitro-N-nitroso-guanidine (MNNG). Mucosa ***samples*** and organ cultures, respectively, underwent a modified comet assay on days 1, 7 and 11. Genotoxicity could be shown for NDEA, Na2Cr2O7 and MNNG on days 1, 7 and 11. Duration of tissue culture and repetitive incubations did not significantly influence the results for NDEA. Nevertheless, Na2Cr2O7 and MNNG caused higher genotoxic effects on cultures subjected to the comet assay on day 11. This model may help to assess genotoxic hazards posed by environmental pollutants that have a cumulative character in repetitive or chronic exposure in vivo.

CONCEPT CODE: Digestive system - Physiology and biochemistry 14004
Genetics - General 03502
Genetics - Human 03508
Respiratory system - Physiology and biochemistry 16004
Toxicology - General and methods 22501

INDEX TERMS: Major Concepts
Genetics; Toxicology

INDEX TERMS: Parts, Structures, & Systems of Organisms
aerodigestive tract: digestive system; nasal
epithelia: respiratory system, organ
culture

INDEX TERMS: Chemicals & Biochemicals
nitroso compounds: genotoxicity; sodium dichromate:
genotoxicity

INDEX TERMS: Methods & Equipment
comet assay: toxicity testing method

ORGANISM: Classifier
Hominidae 86215
Super Taxa
Primates; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
human
Taxa Notes
Animals, Chordates, Humans, Mammals, Primates,
Vertebrates

REGISTRY NUMBER: 10588-01-9 (sodium dichromate)

L136 ANSWER 40 OF 47 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on
STN DUPLICATE 6

ACCESSION NUMBER: 1998:40981 BIOSIS

DOCUMENT NUMBER: PREV199800040981

TITLE: Comparative analysis of cyto- and genotoxic effects of
airborne particulates on human and rodent
respiratory cells in vitro.

AUTHOR(S): Hornberg, C.; Maciuleviciute, L.; Seemayer, N. H.

CORPORATE SOURCE: Med. Inst. Environ. Hyg., Univ. Duesseldorf, Auf m
Hennekamp 50, D-40225 Duesseldorf, Germany

SOURCE: Toxicology In Vitro, (Oct., 1997) Vol. 11, No. 5, pp.
711-715. print.

CODEN: TIVIEQ. ISSN: 0887-2333.

DOCUMENT TYPE: Article

LANGUAGE: English

ENTRY DATE: Entered STN: 14 Jan 1998

Last Updated on STN: 14 Jan 1998

ABSTRACT: In our highly industrialized world **air pollution**
has become an important topic. Beside gaseous pollutants **airborne**
particulates are of great medical concern, containing several hundred mostly
organic substances. They are incriminated to cause an excess mortality.
Airborne particulates were collected in the heavily industrialized Ruhr
region utilizing a high volume sampler HVS 150 (Strohlein Instruments) equipped
with glass fibre filters. Chemical substances were extracted with
dichloromethane and quantitatively transferred to dimethyl sulfoxide for tissue
culture experiments. Cytotoxicity of extracts was determined by
reduction of 'plating efficiency' of human cell line A-549 (pneumocyte type
II). The induction of 'sister chromatid exchanges' was used as a sensitive
bioassay for detection of genotoxic activity of **airborne**
particulates. As target cells we utilized tracheal epithelial cells of the
Syrian golden hamster and the rat, human bronchial epithelial cells of line
BEAS-2B and human lymphocytes. Quantities of substances equivalent to
airborne particulates from 4 and more ml air exerted cytotoxic effects,
while quantities of substances from 0.5 ml of air were markedly genotoxic.

CONCEPT CODE: Toxicology - General and methods 22501
Cytology - Animal 02506
Genetics - Animal 03506
Respiratory system - Physiology and biochemistry 16004
In vitro cellular and subcellular studies 32600
Respiratory system - General and methods 16001

INDEX TERMS: Major Concepts
Toxicology

INDEX TERMS: Parts, Structures, & Systems of Organisms
tracheal **epithelial** cells: **respiratory**
system

INDEX TERMS: Miscellaneous Descriptors
airborne particulates; cytotoxic effect;
genotoxic effect; sister chromatid exchanges

ORGANISM: Classifier
Cricetidae 86310
Super Taxa
Rodentia; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
Syrian golden hamster
Taxa Notes

ORGANISM: Animals, Chordates, Mammals, Nonhuman Vertebrates,
Nonhuman Mammals, Rodents, Vertebrates
Classifier

Searched by Barb O'Bryen, STIC 2-2518

Hominidae 86215
 Super Taxa
 Primates; Mammalia; Vertebrata; Chordata; Animalia
 Organism Name
 A-549: human pneumocytes
 BEAS-2B: human bronchial epithelial cells
 Taxa Notes
 Animals, Chordates, Humans, Mammals, Primates,
 Vertebrates
 Classifier
 Muridae 86375
 Super Taxa
 Rodentia; Mammalia; Vertebrata; Chordata; Animalia
 Organism Name
 rat
 Taxa Notes
 Animals, Chordates, Mammals, Nonhuman Vertebrates,
 Nonhuman Mammals, Rodents, Vertebrates

ORGANISM:

L136 ANSWER 41 OF 47 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on
 STN
 ACCESSION NUMBER: 2005:186433 BIOSIS
 DOCUMENT NUMBER: PREV200500186977
 TITLE: A new approach to **pharmacological** effects on
 ciliary beat frequency in cell
 cultures-exemplary measurements under Pelargonium sidoides
 extract.(EPs 7630).
 AUTHOR(S): Neugebauer, P. [Reprint Author]; Mickenhagen, A.; Siefer,
 O.; Walger, M.
 CORPORATE SOURCE: ENT Dept, Univ Cologne, Cologne, Germany
 hno.neugebauer@uni-koeln.de
 SOURCE: Phytomedicine (Jena), (January 2005) Vol. 12, No. 1-2, pp.
 46-51. print.
 ISSN: 0944-7113 (ISSN print).
 DOCUMENT TYPE: Article
 LANGUAGE: English
 ENTRY DATE: Entered STN: 18 May 2005
 Last Updated on STN: 18 May 2005
 ABSTRACT: The **ciliary beat** frequency (CBF) is an important
 parameter of the defence mechanism of the mucociliary system. We present a new
 method to determine **pharmacological** effects on CBF in vitro.
 Ciliated cell cultures of human nasal epithelium were obtained from partial
 resection of hyperplastic inferior turbinates in rhinosurgery. An adherent
 monolayer culture of ciliated cells was present after 10 days in vitro. This
 study exemplary examines, if a special extract from the roots of Pelargonium
 sidoides (EPs 7630) has an effect on the CBF in vitro. The influence of three
 concentrations of the extract (1, 30, 100 mug/ml) was tested. EPs 7630
 significantly and concentration-dependently increased CBF to 123% at 30 mug/ml
 and to 133% at 100 mug/ml compared to the equilibration phase (100%). After
 rinsing with extract-free medium the CBF of cultured cells returned to nearly
 the normal range. In future, drug manipulation of the CBF by local application
 of rhinologics could be a new therapeutical concept in the treatment of upper
 airway diseases. Copyright 2004 Elsevier GmbH. All rights reserved.
 CONCEPT CODE: Pathology - Therapy 12512
 Respiratory system - Physiology and biochemistry 16004
 Respiratory system - Pathology 16006
 Sense organs - Physiology and biochemistry 20004
 Pharmacology - Clinical pharmacology 22005
 Pharmacology - Immunological processes and allergy 22018
 Immunology - General and methods 34502

Chemotherapy - General, methods and metabolism 38502
 Chemotherapy - Antibacterial agents 38504
 Plant physiology - Respiration, fermentation 51508
 Pharmacognosy and pharmaceutical botany 54000

INDEX TERMS: Major Concepts
 Immune System (Chemical Coordination and Homeostasis);
 Pharmacognosy (Pharmacology); Respiratory System
 (Respiration); Sense Organs (**Sensory**
 Reception)

INDEX TERMS: Parts, Structures, & Systems of Organisms
 nasal **epithelium: respiratory**
 system, **sensory** system

INDEX TERMS: Diseases
 upper airway disease: respiratory system disease, drug
 therapy

INDEX TERMS: Chemicals & Biochemicals
 Pelargonium sidoides root extract: antibacterial-drug,
 antiinfective-drug, immunologic-drug

INDEX TERMS: Miscellaneous Descriptors
ciliary beat frequency;
pharmacological effect

ORGANISM: Classifier
 Geraniaceae 26105
 Super Taxa
 Dicotyledones; Angiospermae; Spermatophyta; Plantae
 Organism Name
 Pelargonium sidoides (species)
 Taxa Notes
 Angiosperms, Dicots, Plants, Spermatophytes, Vascular
 Plants

ORGANISM: Classifier
 Hominidae 86215
 Super Taxa
 Primates; Mammalia; Vertebrata; Chordata; Animalia
 Organism Name
 human (common)
 Taxa Notes
 Animals, Chordates, Humans, Mammals, Primates,
 Vertebrates

L136 ANSWER 42 OF 47 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on
 STN

ACCESSION NUMBER: 2001:109534 BIOSIS
 DOCUMENT NUMBER: PREV200100109534
 TITLE: Neurogenic influence in inflammatory sensitivity to airway
 pollutants.

AUTHOR(S): Roy, J. [Reprint author]; Oortgiesen, M.; Carter, J. D.;
 Boyes, W. K.; Veronesi, B.

CORPORATE SOURCE: National Research Council, Washington, DC, USA
 SOURCE: Society for Neuroscience Abstracts, (2000) Vol. 26, No.
 1-2, pp. Abstract No.-719.11. print.
 Meeting Info.: 30th Annual Meeting of the Society of
 Neuroscience. New Orleans, LA, USA. November 04-09, 2000.
 Society for Neuroscience.
 ISSN: 0190-5295.

DOCUMENT TYPE: Conference; (Meeting)
 Conference; Abstract; (Meeting Abstract)

LANGUAGE: English

ENTRY DATE: Entered STN: 28 Feb 2001
 Last Updated on STN: 15 Feb 2002

ABSTRACT: Particulate matter (PM) is a major **air pollutant** that has been associated with respiratory disorders in susceptible populations. Previous work has demonstrated that PM activates **sensory** receptors (i.e. capsaicin, pH sensitive) found on nerve fibers that innervate the airways and on bronchial epithelial cells. Activation of these receptors initiates a process known as neurogenic inflammation which results in neuropeptide and cytokine release. Previous experiments report that the BALB/c mouse strain is responsive to PM-inflammation in contrast to the non-responsive C57/blk (B6) mouse strain. This differential sensitivity is retained in **sensory** neurons **cultured** from dorsal root ganglia (DRG) and trigeminal ganglia (TG), since neurons from BALB/c **sensory** ganglia release higher levels of inflammatory cytokines in response to PM and other irritants (acid pH, capsaicin) relative to B6 neurons. In the present study, we use RT-PCR, cobalt histochemistry and immunocytochemical techniques to show that the expression of capsaicin (VR-1) and Substance P (NK-1) receptors and release of inflammatory cytokines and neuropeptides are higher in **sensory** neurons from BALB/c strain relative to B6. In conclusion, strain-specific inflammation to PM and other irritants appears to be mediated by the population of irritant and neuropeptide receptors found on DRG and TG neurons. J. Roy holds a National Research Council research associate award at the National Health and Environmental Effects Research Laboratory, N.C.

CONCEPT CODE: Cytology - Animal 02506
 General biology - Symposia, transactions and proceedings 00520
 Biochemistry studies - Nucleic acids, purines and pyrimidines 10062
 Biochemistry studies - Proteins, peptides and amino acids 10064
 Respiratory system - Physiology and biochemistry 16004
 Respiratory system - Pathology 16006
 Nervous system - Physiology and biochemistry 20504
 Nervous system - Pathology 20506
 Immunology - Immunopathology, tissue immunology 34508
INDEX TERMS: Major Concepts
 Nervous System (Neural Coordination); Respiratory System (Respiration)
INDEX TERMS: Parts, Structures, & Systems of Organisms
 airway: respiratory system; bronchial **epithelial** cells: **respiratory** system; dorsal root ganglia: nervous system; **sensory** neurons: nervous system; trigeminal ganglia: nervous system
INDEX TERMS: Diseases
 neurogenic inflammation: immune system disease, nervous system disease
INDEX TERMS: Diseases
 respiratory disorder: respiratory system disease
INDEX TERMS: Chemicals & Biochemicals
 capsaicin receptors [VR-1 receptor]: expression; inflammatory cytokines; neuropeptides; particulate matter: **air pollutant**, inflammatory sensitivity; substance P receptor [NK-1 receptor]: expression
INDEX TERMS: Methods & Equipment
 cobalt histochemistry: analytical method; immunocytochemical techniques: analytical method; reverse transcription-polymerase chain reaction: analytical method
INDEX TERMS: Miscellaneous Descriptors
 Meeting Abstract
ORGANISM: Classifier

Muridae 86375
 Super Taxa
 Rodentia; Mammalia; Vertebrata; Chordata; Animalia
 Organism Name
 mouse: BALB/c, C57/B6
 Taxa Notes
 Animals, Chordates, Mammals, Nonhuman Vertebrates,
 Nonhuman Mammals, Rodents, Vertebrates

L136 ANSWER 43 OF 47 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on
 STN

ACCESSION NUMBER: 1977:168973 BIOSIS
 DOCUMENT NUMBER: PREV197763063837; BA63:63837
 TITLE: STUDIES ON CYSTIC FIBROSIS USING ISO ELECTRIC FOCUSING PART
 3 CORRELATION BETWEEN CYSTIC FIBROSIS PROTEIN AND CILIARY
 DYS KINESIA ACTIVITY IN SERUM SHOWN BY A MODIFIED RABBIT
 TRACHEAL BIOASSAY.
 AUTHOR(S): WILSON G B; MOSHER M T; FUDENBERG H H
 SOURCE: Pediatric Research, (1977) Vol. 11, No. 2, pp. 143-146.
 CODEN: PEREBL. ISSN: 0031-3998.

DOCUMENT TYPE: Article
 FILE SEGMENT: BA
 LANGUAGE: Unavailable

ABSTRACT: A modified rabbit tracheal **bioassay** was developed for use in investigating a possible correlation between cystic fibrosis protein (CFP) and ciliary dyskinesia factor (CDF) in human serum. The **bioassay** requires high standards of tissue selection, and all epithelial tissue must be free of underlying connective tissue. When serum **samples** were collected and processed carefully and warmed to 37° before assay, CDF could be reliably detected in 31 of 31 sera from cystic fibrosis (CF) homozygotes or obligate heterozygotes in 35 min or less without prior fractionation or connection of sera, whereas 13 of 14 normal control sera were nonreactive. CDF-positive serum reacts in 3 consecutive phases: initial increase in **ciliary beat** frequency, **ciliary** dyskinesia and tissue destruction with extrusion of single ciliated cells, mucus and debris. These results confirm the association of CDF with cystic fibrosis. The **bioassay** is not specific for CF when whole sera are *****bioassayed*****, since serum from several patients with bronchial asthma also caused ciliary dyskinesia. This finding need not preclude using rabbit tracheal **ciliated epithelial** tissue as an assay for following the purification of CDF. Isoelectric focusing showed that the presence or absence of CFP corresponded with that of dyskinesia activity in all sera tested except for the active **samples** from 7 asthma patients, which were negative for CFP. CFP and CDF may be identical or closely related markers for the CF gene. The activity detected by the rabbit tracheal *****bioassay***** in sera from patients with asthma and other diseases probably is caused by a substance different from a CF-specific CDF. Several substances in human serum can produce ciliary dyskinesia in rabbit tracheal epithelium, but only 1 appears to be a specific marker for CF. CFP may be related structurally or metabolically to a CF-specific CDF. Patients with bronchial asthma and other respiratory and autoimmune disorders may harbor a substance that can produce ciliary dyskinesia but that differs from CF-specific CDF.

CONCEPT CODE: Genetics - Human 03508
 Clinical biochemistry - General methods and applications
 10006
 Biochemistry methods - Proteins, peptides and amino acids
 10054
 Biochemistry studies - Proteins, peptides and amino acids
 10064
 Biophysics - Methods and techniques 10504

Movement 12100
Pathology - Inflammation and inflammatory disease 12508
Metabolism - Metabolic disorders 13020
Blood - Blood and lymph studies 15002
Respiratory system - Pathology 16006
Development and Embryology - Pathology 25503
In vitro cellular and subcellular studies 32600
Immunology - Immunopathology, tissue immunology 34508
Allergy 35500

INDEX TERMS: Major Concepts
Biochemistry and Molecular Biophysics; Blood and
Lymphatics (Transport and Circulation); Clinical
Chemistry (Allied Medical Sciences); Genetics;
Metabolism; Pulmonary Medicine (Human Medicine, Medical
Sciences)

INDEX TERMS: Miscellaneous Descriptors
HUMAN BRONCHIAL ASTHMA

ORGANISM: Classifier
Leporidae 86040
Super Taxa
Lagomorpha; Mammalia; Vertebrata; Chordata; Animalia
Taxa Notes
Animals, Chordates, Lagomorphs, Mammals, Nonhuman
Vertebrates, Nonhuman Mammals, Vertebrates

ORGANISM: Classifier
Hominidae 86215
Super Taxa
Primates; Mammalia; Vertebrata; Chordata; Animalia
Taxa Notes
Animals, Chordates, Humans, Mammals, Primates,
Vertebrates

L136 ANSWER 44 OF 47 TOXCENTER COPYRIGHT 2005 ACS on STN DUPLICATE 4
ACCESSION NUMBER: 2000:8235 TOXCENTER
DOCUMENT NUMBER: PubMed ID: 10564181
TITLE: Effects of aqueous extracts of PM(10) filters from the
Utah valley on human airway epithelial cells
AUTHOR(S): Frampton M W; Ghio A J; Samet J M; Carson J L; Carter J D;
Devlin R B
CORPORATE SOURCE: Department of Medicine, University of Rochester School of
Medicine and Dentistry, Rochester, New York 14642, USA
CONTRACT NUMBER: R01-ES-02679 (NIEHS)
R01-HL-51701 (NHLBI)
SOURCE: American journal of physiology, (1999 Nov) 277 (5 Pt 1)
L960-7.
Journal Code: 0370511. ISSN: 0002-9513.
COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
FILE SEGMENT: MEDLINE
OTHER SOURCE: MEDLINE 2000035097
LANGUAGE: English
ENTRY DATE: Entered STN: 20011116
Last Updated on STN: 20011116

ABSTRACT:
We hypothesized that the reduction in hospital respiratory admissions in the
Utah Valley during closure of a local steel mill in 1986-1987 was attributable
in part to decreased toxicity of ambient air particles. **Sampling**
filters for particulate matter < 10 micrometer (PM(10)) were obtained from a
Utah Valley monitoring station for the year before (year 1), during (year 2),
and after (year 3) the steel mill closure. Aqueous extracts of the filters

were analyzed for metal content and oxidant production and added to ***cultures*** of human **respiratory epithelial** (BEAS-2B) cells for 2 or 24 h. Year 2 dust contained the lowest concentrations of soluble iron, copper, and zinc and showed the least oxidant generation. Only dust from year 3 caused cytotoxicity (by microscopy and lactate dehydrogenase release) at 500 microgram/ml. Year 1 and year 3, but not year 2, dust induced expression of interleukin-6 and -8 in a dose-response fashion. The effects of ambient air particles on human respiratory epithelial cells vary significantly with time and metal concentrations.

CONTROLLED TERM: Check Tags: Comparative Study
*Air Pollutants, Occupational: TO, toxicity
Cytotoxins: PD, pharmacology
*Epithelial Cells: DE, drug effects
Epithelial Cells: IM, immunology
Epithelial Cells: UL, ultrastructure
Filtration
Gene Expression: DE, drug effects
Gene Expression: IM, immunology
Humans
Interleukin-6: GE, genetics
Interleukin-6: IM, immunology
Interleukin-8: GE, genetics
Interleukin-8: IM, immunology
*Lung: CY, cytology
Metals, Heavy: PD, pharmacology
Microscopy, Electron, Scanning
Occupational Diseases: CI, chemically induced
Occupational Diseases: IM, immunology
Occupational Diseases: PA, pathology
Oxidative Stress: DE, drug effects
RNA, Messenger: AN, analysis
Research Support, U.S. Gov't, P.H.S.
*Steel
Utah
Ventilation
Water
REGISTRY NUMBER: 12597-69-2 (Steel)
7732-18-5 (Water)
CHEMICAL NAME: 0 (Air Pollutants, Occupational); 0
(Cytotoxins); 0 (Interleukin-6); 0 (Interleukin-8); 0
(Metals, Heavy); 0 (RNA, Messenger)
L136 ANSWER 45 OF 47 TOXCENTER COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2004:325342 TOXCENTER
DOCUMENT NUMBER: CRISP-2003-ES009844-04
TITLE: **SENSORY IRRITANT RECEPTORS AND PARTICULATE INFLAMMATION**
AUTHOR(S): SIMON S A
CORPORATE SOURCE: SAS@NEURO.DUKE.EDU, DUKE UNIVERSITY MEDICAL CENTER, BOX
3209, DURHAM, NC 27710:NORTH CAROLINA
SUPPORTING ORGANIZATION (SPONSORING AGENCY): U.S. DEPT. OF HEALTH AND HUMAN
SERVICES; PUBLIC HEALTH SERVICE; NATIONAL INSTITUTES OF
HEALTH, NATIONAL INSTITUTE OF ENVIRONMENTAL HEALTH
SCIENCES
SOURCE: Crisp Data Base National Institutes of Health.
DOCUMENT TYPE: (Research)
FILE SEGMENT: CRISP
LANGUAGE: English
ENTRY DATE: Entered STN: 20041229

Last Updated on STN: 20041229

ABSTRACT:

Increases in pulmonary related incidents of morbidity and mortality have been epidemiologically associated with exposure to particulate matter (PM). Due to its heterogeneity and complex physicochemistry, the underlying mechanism(s) of PM toxicity are poorly understood. This proposal examines the hypothesis that physicochemical components of PM initiate inflammation by activation of irritant (i.e., capsaicin, acid-sensitive) receptors located on **sensory** nerve endings and airway epithelial target cells. This activation results in an influx of extracellular calcium and release of neuropeptides and inflammatory cytokines, which proceed to initiate and sustain events of airway inflammation and hypersensitivity. The response of irritant receptors to PM will be examined in primary **cultures** of **sensory** dorsal root ganglion neurons and in human bronchial epithelial cells. Biophysical (e.g., patch clamp, calcium imaging) and immunological (e.g., cytokine release) endpoints will be used to characterize the response to various urban, industrial and ambient PM. The physicochemical properties of selected PM and their separate soluble and particulate fractions will be determined and examined for their cellular effects. Based on these data, synthetic particle analogues will be designed, that resemble PM particles in size and surface charge, to assess the inflammatory contribution of specific PM components. Physiological and pharmacological evaluation of the responses to PM will identify and characterize the contribution of culpable irritant receptors to the PM-mediated effects. In addition, the involvement of other cellular pathways (i.e., calcium channels, acid-sensitive exchangers) will be examined. The mechanistic approach outlined in this proposal will identify the responsible factors and target sites contributing to PM toxicity.

SUPPLEMENTARY TERMS: Miscellaneous Descriptors

air pollution; laboratory mouse;
inflammation; irritation, irritant; pharmacokinetics;
environmental health; environmental contamination; spinal
ganglion; particle; **respiratory**
epithelium; tissue, cell culture;
environmental toxicology; **sensory** receptor

L136 ANSWER 46 OF 47 TOXCENTER COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2003:156403 TOXCENTER

DOCUMENT NUMBER: CRISP-2002-ES09844-03

TITLE: **SENSORY** IRRITANT RECEPTORS AND PARTICULATE
INFLAMMATION

AUTHOR(S): SIMON S A

CORPORATE SOURCE: SAS@NEURO.DUKE.EDU, DUKE UNIVERSITY MEDICAL CENTER, BOX
3209, DURHAM, NC 27710:NORTH CAROLINASUPPORTING ORGANIZATION (SPONSORING AGENCY): U.S. DEPT. OF HEALTH AND HUMAN
SERVICES; PUBLIC HEALTH SERVICE; NATIONAL INSTITUTES OF
HEALTH, NATIONAL INSTITUTE OF ENVIRONMENTAL HEALTH
SCIENCES

SOURCE: Crisp Data Base National Institutes of Health.

DOCUMENT TYPE: (Research)

FILE SEGMENT: CRISP

LANGUAGE: English

ENTRY DATE: Entered STN: 20030708

Last Updated on STN: 20030708

ABSTRACT:

Increases in pulmonary related incidents of morbidity and mortality have been epidemiologically associated with exposure to particulate matter (PM). Due to its heterogeneity and complex physicochemistry, the underlying mechanism(s) of PM toxicity are poorly understood. This proposal examines the hypothesis that physicochemical components of PM initiate inflammation by activation of irritant (i.e., capsaicin, acid-sensitive) receptors located on **sensory**

Searched by Barb O'Bryen, STIC 2-2518

nerve endings and airway epithelial target cells. This activation results in an influx of extracellular calcium and release of neuropeptides and inflammatory cytokines, which proceed to initiate and sustain events of airway inflammation and hypersensitivity. The response of irritant receptors to PM will be examined in primary cultures of sensory dorsal root ganglion neurons and in human bronchial epithelial cells. Biophysical (e.g., patch clamp, calcium imaging) and immunological (e.g., cytokine release) endpoints will be used to characterize the response to various urban, industrial and ambient PM. The physicochemical properties of selected PM and their separate soluble and particulate fractions will be determined and examined for their cellular effects. Based on these data, synthetic particle analogues will be designed, that resemble PM particles in size and surface charge, to assess the inflammatory contribution of specific PM components. Physiological and pharmacological evaluation of the responses to PM will identify and characterize the contribution of culpable irritant receptors to the PM-mediated effects. In addition, the involvement of other cellular pathways (i.e., calcium channels, acid-sensitive exchangers) will be examined. The mechanistic approach outlined in this proposal will identify the responsible factors and target sites contributing to PM toxicity.

SUPPLEMENTARY TERMS: Miscellaneous Descriptors
 air pollution; laboratory mouse;
 inflammation; irritation, irritant; pharmacokinetics;
 environmental health; environmental contamination; spinal
 ganglion; particle; respiratory
 epithelium; tissue, cell culture;
 environmental toxicology; sensory receptor

L136 ANSWER 47 OF 47 TOXCENTER COPYRIGHT 2005 ACS on STN
 ACCESSION NUMBER: 2002:559667 TOXCENTER
 DOCUMENT NUMBER: CRISP-2000-ES09844-01A2
 TITLE: SENSORY IRRITANT RECEPTORS AND PARTICULATE
 INFLAMMATION
 AUTHOR(S): SIMON S A
 CORPORATE SOURCE: DUKE UNIVERSITY MEDICAL CENTER, BOX 3209, DURHAM, NC
 27710:NORTH CAROLINA
 SUPPORTING ORGANIZATION (SPONSORING AGENCY): U.S. DEPT. OF HEALTH AND HUMAN
 SERVICES; PUBLIC HEALTH SERVICE; NATIONAL INSTITUTES OF
 HEALTH, NATIONAL INSTITUTE OF ENVIRONMENTAL HEALTH
 SCIENCES
 SOURCE: Crisp Data Base National Institutes of Health.
 DOCUMENT TYPE: (Research)
 FILE SEGMENT: CRISP
 LANGUAGE: English
 ENTRY DATE: Entered STN: 20021200
 Last Updated on STN: 20021200

ABSTRACT:
 Increases in pulmonary related incidents of morbidity and mortality have been epidemiologically associated with exposure to particulate matter (PM). Due to its heterogeneity and complex physicochemistry, the underlying mechanism(s) of PM toxicity are poorly understood. This proposal examines the hypothesis that physicochemical components of PM initiate inflammation by activation of irritant (i.e., capsaicin, acid-sensitive) receptors located on nerve endings and airway epithelial target cells. This activation results in an influx of extracellular calcium and release of neuropeptides and inflammatory cytokines, which proceed to initiate and sustain events of airway inflammation and hypersensitivity. The response of irritant receptors to PM will be examined in primary cultures of sensory dorsal root ganglion neurons and in human bronchial epithelial cells. Biophysical (e.g., patch clamp, calcium imaging) and immunological (e.g., cytokine release) endpoints will be used to characterize the response

to various urban, industrial and ambient PM. The physicochemical properties of selected PM and their separate soluble and particulate fractions will be determined and examined for their cellular effects. Based on these data, synthetic particle analogues will be designed, that resemble PM particles in size and surface charge, to assess the inflammatory contribution of specific PM components. Physiological and pharmacological evaluation of the responses to PM will identify and characterize the contribution of culpable irritant receptors to the PM-mediated effects. In addition, the involvement of other cellular pathways (i.e., calcium channels, acid-sensitive exchangers) will be examined. The mechanistic approach outlined in this proposal will identify the responsible factors and target sites contributing to PM toxicity.

SUPPLEMENTARY TERMS: Miscellaneous Descriptors

air pollution; laboratory mouse;
inflammation; irritation, irritant; pharmacokinetics;
environmental health; environmental contamination; spinal
ganglion; particle; respiratory
epithelium; tissue, cell culture;
environmental toxicology; sensory receptor

FILE 'HOME' ENTERED AT 16:11:16 ON 16 DEC 2005

=>

Searched by Barb O'Bryen, STIC 2-2518

=> d his nofile

(FILE 'HOME' ENTERED AT 14:50:08 ON 16 DEC 2005)

FILE 'CAPLUS' ENTERED AT 14:50:30 ON 16 DEC 2005

```

SET LINE 250
SET DETAIL OFF
E US2004-798986/AP,PRN 25
SET LINE LOGIN
SET DETAIL LOGIN
L1      493 SEA ABB=ON CHIN W?/AU
L2      2192 SEA ABB=ON KWON S?/AU
L3      0 SEA ABB=ON L1 AND L2
        E BIOSENSOR/CT
        E E4+ALL
L4      16036 SEA ABB=ON BIOSENSORS/CT
L5      2 SEA ABB=ON (L1 OR L2) AND L4
        D SCAN
L6      3734 SEA ABB=ON EPITHELI?/OBI (L) (RESPIRATORY/OBI OR CILIAT?/OBI OR
        GOBLET?/OBI)
L7      1 SEA ABB=ON L4 AND L6
        D SCAN
        E ENVIRONMENTAL POLLUTION/CT
        E E3+ALL
L8      208880 SEA ABB=ON CULTUR?/OBI
L9      244 SEA ABB=ON L6 AND L8
L10     42474 SEA ABB=ON ENVIRONMENTAL POLLUTION/CT
L11     147315 SEA ABB=ON AIR/OBI (L) POLLUTION/CW
L12     4705 SEA ABB=ON (BIOLOGICAL/OBI OR CHEMICAL/OBI) (L) WARFARE/OBI
L13     13214 SEA ABB=ON AIR/OBI (L) MONITOR?/OBI
L14     11081 SEA ABB=ON SAMPLING/CT
L15     4 SEA ABB=ON L9 AND (L10 OR L11 OR L12 OR L13 OR L14)
        D SCAN
L16     11248 SEA ABB=ON AIRBORNE PARTICLES/CT
L17     56518 SEA ABB=ON TOXICITY/CT
L18     4102 SEA ABB=ON ECOTOXICITY/CT
L19     9069 SEA ABB=ON BIOASSAY/CT
L20     427058 SEA ABB=ON 59/SC, SX
L21     615834 SEA ABB=ON 4/SC, SX
L22     14 SEA ABB=ON L6 AND L19
L23     3 SEA ABB=ON L22 AND ((L10 OR L11 OR L12 OR L13 OR L14) OR (L16
        OR L17 OR L18))
L24     124 SEA ABB=ON L8 AND L20 AND L21
L25     74 SEA ABB=ON L8 AND L20 AND L21 AND ((L10 OR L11 OR L12 OR L13)
        OR L16)
L26     20819 SEA ABB=ON SAMPLING/CW
L27     4 SEA ABB=ON L9 AND ((L10 OR L11 OR L12 OR L13) OR L26)
L28     3 SEA ABB=ON L22 AND ((L10 OR L11 OR L12 OR L13) OR L26 OR (L16
        OR L17 OR L18))
L29     3 SEA ABB=ON L25 AND L26
L30     3 SEA ABB=ON L29 NOT (L7 OR L27 OR L28)
        D SCAN TI
L31     647 SEA ABB=ON (CILIA? (2A) BEAT?)/BI
L32     2600 SEA ABB=ON (ELECTRICAL RESPON?)/BI
L33     1144 SEA ABB=ON (SECRET? (2A) MUCIN#)/BI
L34     28 SEA ABB=ON L6 AND (L31 OR L32 OR L33) AND (L4 OR (L10 OR L11
        OR L12 OR L13) OR (L16 OR L17 OR L18 OR L19 OR L20 OR L21) OR
        L26)
        D QUE
L35     9 SEA ABB=ON L6 AND (L31 OR L32 OR L33) AND (L4 OR (L10 OR L11

```

OR L12 OR L13) OR (L16 OR L17 OR L18 OR L19 OR L20) OR L26)
 D SCAN TI
 L36 3 SEA ABB=ON VITRO/TI AND L35
 D SCAN
 L37 8 SEA ABB=ON L6 AND (L31 OR L32 OR L33) AND (L4 OR (L10 OR L11
 OR L12 OR L13) OR (L16 OR L17 OR L18 OR L19 OR L20) OR L26)
 AND L21

INDEX '1MOBILITY, 2MOBILITY, ABI-INFORM, ADISCTI, AEROSPACE, AGRICOLA,
 ALUMINIUM, ANABSTR, ANTE, APOLLIT, AQUALINE, AQUASCI, AQUIRE, BABS,
 BIBLIODATA, BIOBUSINESS, BIOCOMMERCE, BIOENG, BIOSIS, BIOTECHABS,
 BIOTECHDS, BIOTECHNO, BLLDB, CABA, CANCERLIT, ...' ENTERED AT 15:10:49 ON
 16 DEC 2005

SEA (AIR(2A) (POLLUTION OR QUALITY)) OR AIRBORNE OR ((BIOLOGICAL

 2357 FILE 1MOBILITY
 81 FILE 2MOBILITY
 13485 FILE ABI-INFORM
 298 FILE ADISCTI
 51582 FILE AEROSPACE
 12003 FILE AGRICOLA
 931 FILE ALUMINIUM
 2212 FILE ANABSTR
 2308 FILE ANTE
 106 FILE APOLLIT
 2064 FILE AQUALINE
 5982 FILE AQUASCI
 19 FILE AQUIRE
 1154 FILE BABS
 256 FILE BIBLIODATA
 16668 FILE BIOBUSINESS
 143 FILE BIOCOMMERCE
 3018 FILE BIOENG
 45230 FILE BIOSIS
 299 FILE BIOTECHABS
 299 FILE BIOTECHDS
 4090 FILE BIOTECHNO
 26656 FILE CABA
 2070 FILE CANCERLIT
 1005 FILE CAOLD
 171800 FILE CAPLUS
 88 FILE CASREACT
 2471 FILE CBNB
 6473 FILE CEABA-VTB
 898 FILE CEN
 798 FILE CERAB
 4901 FILE CIN
 6092 FILE CIVILENG
 72364 FILE COMPENDEX
 1817 FILE COMPUAB
 224 FILE COMPUSCIENCE
 4698 FILE CONFSCI
 575 FILE COPPERLIT
 310 FILE CORROSION
 136 FILE CROPB
 394 FILE CROPU
 2869 FILE CSNB
 132 FILE DDFB
 67 FILE DDFU
 2 FILE DETHERM

3354 FILE DGENE
3491 FILE DISSABS
199 FILE DKF
2083 FILE DPCI
132 FILE DRUGB
122 FILE DRUGU
1765 FILE ELCOM
279 FILE EMA
270 FILE EMBAL
53194 FILE EMBASE
46972 FILE ENCOMPLIT
7058 FILE ENCOMPPAT
157337 FILE ENERGY
20627 FILE ENTEC
15699 FILE ENVIROENG
6514 FILE EPFULL
11375 FILE ESBIODBASE
1 FILE FOMAD
7 FILE FORIS
134 FILE FRANCEPAT
529 FILE FRFULL
656 FILE FROSTI
753 FILE FSTA
2691 FILE GBFULL
705 FILE GENBANK
19183 FILE GEOREF
8597 FILE HEALSAFE
5329 FILE ICONDA
3 FILE IFICLS
7721 FILE IFIPAT
12 FILE IMSDRUGNEWS
23 FILE INFODATA
21605 FILE INIS
5150 FILE INPADO
46245 FILE INSPEC
1726 FILE INSPHYS
31300 FILE INVESTEXT
409 FILE IPA
8635 FILE ITRD
1753 FILE JAPIO
27982 FILE JICST-EPLUS
613 FILE KOREAPAT
116 FILE KOSMET
10472 FILE LIFESCI
96 FILE LISA
920 FILE MATBUS
425 FILE MATH
12356 FILE MECHENG
35790 FILE MEDLINE
2588 FILE METADEX
14 FILE NAPRALERT
16557 FILE NIOSHTIC
46175 FILE NLDB
82731 FILE NTIS
4 FILE NUTRACEUT
2630 FILE OCEAN
4669 FILE PAPERCHEM2
78743 FILE PASCAL
4 FILE PATDPA
246 FILE PATDPAFULL

11213 FILE PCTFULL
 18 FILE PHARMAML
 326 FILE PHIN
 2542 FILE PIRA ,
 38053 FILE POLLUAB
 76931 FILE PROMT
 2251 FILE RAPRA
 5643 FILE RSWB
 287 FILE RUSSIAPAT
 39834 FILE SCISEARCH
 539 FILE SOLIDSTATE
 203 FILE SOLIS
 10988 FILE TEMA
 2481 FILE TEXTILETECH
 149730 FILE TOXCENTER
 73 FILE TRIBO
 7341 FILE TULSA
 6609 FILE TULSA2
 4561 FILE UFORDAT
 36533 FILE ULIDAT
 46618 FILE USPATFULL
 3917 FILE USPAT2
 23 FILE VETB
 42 FILE VETU
 8695 FILE WATER
 438 FILE WELDASEARCH
 12737 FILE WPIDS
 55 FILE WPIFV
 12737 FILE WPINDEX
 2497 FILE WSCA
 906 FILE WTEXTILES

L38 QUE ABB=ON (AIR(2A) (POLLUTION OR QUALITY)) OR AIRBORNE OR
 ((BIOLOGICAL OR CHEMICAL) (2A) WARFARE)

 D RANK

FILE 'STNGUIDE' ENTERED AT 15:14:17 ON 16 DEC 2005

L39 0 SEA ABB=ON CHIN W?/AU
 L40 0 SEA ABB=ON KWON S?/AU
 L41 0 SEA ABB=ON BIOSENSOR# OR SENSOR#
 L42 0 SEA ABB=ON BIOASSAY?
 L43 3 SEA ABB=ON CULTUR?
 L44 13 SEA ABB=ON (ENVIRONMENT? OR AIR) (2A) (POLLUT? OR MONITOR? OR
 QUALITY)
 L45 0 SEA ABB=ON AIRBORNE OR AIR BORNE
 L46 0 SEA ABB=ON (BIOLOGICAL OR CHEMICAL) (2A) WARFARE
 L47 0 SEA ABB=ON (CILIA? (2A) BEAT?)
 L48 0 SEA ABB=ON (ELECTRICAL RESPONS?)
 L49 0 SEA ABB=ON (SECRET? (2A) MUCIN#)

FILE 'JICST-EPLUS, PASCAL, CABA, BIOTECHNO, ESBIODASE, NTIS, NIOSHTIC,
 ENVIROENG, HEALSAFE, INSPEC, BIOSIS, CONFSCI, LIFESCI, POLLUAB,
 TOXCENTER, CEABA-VTB, WPIX, SCISEARCH' ENTERED AT 15:23:02 ON 16 DEC 2005

L50 3121 SEA ABB=ON L1
 L51 7974 SEA ABB=ON L2
 L52 2197473 SEA ABB=ON BIOSENSOR# OR SENSOR#
 L53 290424 SEA ABB=ON BIOASSAY?
 L54 29232 SEA ABB=ON EPITHELI? (3A) (RESPIRATORY OR CILIAT? OR GOBLET?)
 L55 3624943 SEA ABB=ON CULTUR?
 L56 955407 SEA ABB=ON (ENVIRONMENT? OR AIR) (2A) (POLLUT? OR MONITOR? OR

Searched by Barb O'Bryen, STIC 2-2518

QUALITY)

L57 158030 SEA ABB=ON AIRBORNE OR AIR BORNE
 L58 17285 SEA ABB=ON (BIOLOGICAL OR CHEMICAL) (2A) WARFARE
 L59 4285 SEA ABB=ON (CILIA? (2A) BEAT?)
 L60 7049 SEA ABB=ON (ELECTRICAL RESPONS?)
 L61 4997 SEA ABB=ON (SECRET? (2A) MUCIN#)
 L62 2 SEA ABB=ON L50 AND L51
 D SCAN
 L63 0 SEA ABB=ON (L50 OR L51) AND (L52 OR L53) AND (L54 OR (L56 OR L57 OR L58))
 D KWIC L62
 L64 1378 SEA ABB=ON (L52 OR L53) AND L54
 L65 122 SEA ABB=ON (L52 OR L53) AND L54 AND L55
 L66 16 SEA ABB=ON L65 AND (L56 OR L57 OR L58)
 L67 2 SEA ABB=ON L54 (5A) L55 AND (L56 OR L57 OR L58) AND (L59 OR L60 OR L61)
 L68 1 SEA ABB=ON L64 AND (L56 OR L57 OR L58) AND (L59 OR L60 OR L61)
 L69 32 SEA ABB=ON L64 AND (L59 OR L60 OR L61)
 L70 25 DUP REM L69 (7 DUPLICATES REMOVED)
 ANSWERS '1-3' FROM FILE JICST-EPLUS
 ANSWERS '4-6' FROM FILE PASCAL
 ANSWER '7' FROM FILE BIOTECHNO
 ANSWERS '8-9' FROM FILE ESBIODASE
 ANSWERS '10-11' FROM FILE INSPEC
 ANSWERS '12-23' FROM FILE BIOSIS
 ANSWERS '24-25' FROM FILE SCISEARCH
 D SCAN
 L71 1500078 SEA ABB=ON TOXICITY/CT OR PHARMACOLOGICAL OR SCREEN?/TI
 L72 4696113 SEA ABB=ON SAMPL?
 L73 9 SEA ABB=ON L69 AND (L71 OR L72)
 L74 6 SEA ABB=ON L54 (5A) L55 AND L72 AND (L56 OR L57 OR L58)
 D SCAN
 L75 36347 SEA ABB=ON UTAH
 L76 4 SEA ABB=ON L74 AND L75
 D KWIC
 FILE 'MEDLINE' ENTERED AT 15:38:26 ON 16 DEC 2005
 L77 447 SEA ABB=ON CHIN W?/AU
 L78 540 SEA ABB=ON KWON S?/AU
 L79 0 SEA ABB=ON L77 AND L78
 FILE 'STNGUIDE' ENTERED AT 15:38:40 ON 16 DEC 2005
 FILE 'MEDLINE' ENTERED AT 15:39:31 ON 16 DEC 2005
 L80 3896 SEA ABB=ON BIOLOGICAL WARFARE+NT/CT
 L81 716 SEA ABB=ON CHEMICAL WARFARE+NT/CT
 FILE 'STNGUIDE' ENTERED AT 15:40:38 ON 16 DEC 2005
 FILE 'MEDLINE' ENTERED AT 15:40:46 ON 16 DEC 2005
 L82 102328 SEA ABB=ON ENVIRONMENTAL EXPOSURE+NT/CT
 L83 41512 SEA ABB=ON AIR POLLUTION+NT/CT
 FILE 'STNGUIDE' ENTERED AT 15:42:22 ON 16 DEC 2005
 FILE 'MEDLINE' ENTERED AT 15:42:30 ON 16 DEC 2005
 L84 176076 SEA ABB=ON EPITHELIAL CELLS+NT/CT
 L85 174367 SEA ABB=ON EPITHELIUM+NT/CT
 L86 269379 SEA ABB=ON RESPIRATORY SYSTEM+NT/CT

FILE 'STNGUIDE' ENTERED AT 15:43:47 ON 16 DEC 2005

FILE 'MEDLINE' ENTERED AT 15:43:57 ON 16 DEC 2005

L87 7060 SEA ABB=ON CILIA/CT
 L88 485 SEA ABB=ON GOBLET CELLS+NT/CT
 L89 10594 SEA ABB=ON MUCINS+NT/CT
 L90 8627 SEA ABB=ON BIOSENSING TECHNIQUES+NT/CT
 L91 6517 SEA ABB=ON BIOSENSORS/CT
 L92 0 SEA ABB=ON L91 NOT L90
 L93 758406 SEA ABB=ON CELLS, CULTURED+NT/CT
 L94 13 SEA ABB=ON (L77 OR L78) AND (L80 OR L81 OR L82 OR L83)
 L95 1 SEA ABB=ON (L77 OR L78) AND (L80 OR L81 OR L82 OR L83) AND
 ((L84 OR L85 OR L86 OR L87 OR L88 OR L89 OR L90) OR L93)
 D SCAN
 D TRIAL
 L96 437 SEA ABB=ON (L80 OR L81 OR L82 OR L83) AND ((L84 OR L85) AND
 L86)
 L97 129 SEA ABB=ON L96 AND L93
 L98 0 SEA ABB=ON L97 AND (L90 OR L91)
 L99 5 SEA ABB=ON L97 AND (L87 OR L88 OR L89)
 D TRIAL 1-5
 E AIR POLLUTANTS, ENVIRONMENTAL+ALL/CT
 L100 60664 SEA ABB=ON AIR POLLUTANTS+NT/CT
 D QUE L99
 L101 9 SEA ABB=ON ((L80 OR L81 OR L82 OR L83) OR L100) AND (L84 OR
 L85) AND L86 AND L93 AND (L87 OR L88 OR L89)

FILE 'EMBASE' ENTERED AT 15:51:55 ON 16 DEC 2005

L102 395 SEA ABB=ON CHIN W?/AU
 L103 476 SEA ABB=ON KWON S?/AU
 D QUE L101
 E BIOLOGICAL WARFARE+ALL/CT
 L104 3037 SEA ABB=ON BIOLOGICAL WARFARE/CT
 E CHEMICAL WARFARE+ALL/CT
 L105 996 SEA ABB=ON CHEMICAL WARFARE/CT
 E ENVIRONMENTAL EXPOSURE+ALL/CT
 L106 22836 SEA ABB=ON ENVIRONMENTAL EXPOSURE/CT
 E AIR POLLUTION+ALL/CT
 L107 84207 SEA ABB=ON "AIR AND AIR RELATED PHENOMENA"+NT/CT
 D QUE L101
 E AIR POLLUTANTS+ALL
 E AIR POLLUTANTS+ALL/CT
 E E2+ALL
 D QUE L101
 E RESPIRATORY EPITH/CT
 E E7+ALL
 L108 3900 SEA ABB=ON RESPIRATORY EPITHELIUM/CT
 E CELLS, CULTURED+ALL/CT
 L109 187739 SEA ABB=ON CELL CULTURE/CT
 E CILIA/CT
 E E3+ALL
 L110 218 SEA ABB=ON EUKARYOTIC FLAGELLUM/CT
 E RESPIRATORY TRACT CIL/CT
 E GOBLET CELLS/CT
 E E3+ALL
 L111 2385 SEA ABB=ON GOBLET CELL/CT
 E MUCIN+ALL/CT
 L112 6530 SEA ABB=ON MUCIN/CT
 D QUE L101

Gitomer 10/798986 search history

D QUE L67
 D QUE L68
 D QUE L73
 D QUE L74
 L128 0 S (L66-L68 OR L73-L74) NOT L62
 FILE 'EMBASE' ENTERED AT 16:08:34 ON 16 DEC 2005
 D QUE L118
 D QUE L123
 D QUE L126
 L128 6 SEA ABB=ON L118 OR L123 OR L126
 FILE 'CAPLUS' ENTERED AT 16:08:36 ON 16 DEC 2005
 D QUE L7
 D QUE L27
 D QUE L28
 D QUE L30
 D QUE L37
 L129 18 SEA ABB=ON (L7 OR L27 OR L28 OR L30 OR L37) NOT L5
 FILE 'MEDLINE' ENTERED AT 16:08:38 ON 16 DEC 2005
 D QUE L101
 L130 9 SEA ABB=ON L101 NOT L95
 FILE 'JICST-EPLUS, PASCAL, CABA, BIOTECHNO, ESBIODBASE, NTIS, NIOSHTIC,
 ENVIROENG, HEALSAFE, INSPEC, BIOSIS, CONFSCI, LIFESCI, POLLUAB,
 TOXCENTER, CEABA-VTB, WPIX, SCISEARCH' ENTERED AT 16:08:50 ON 16 DEC 2005
 D QUE L66
 D QUE L67
 D QUE L68
 D QUE L73
 D QUE L74
 L131 19 SEA ABB=ON (L66 OR L67 OR L68)
 L132 15 SEA ABB=ON (L73 OR L74)
 L133 19 SEA ABB=ON L131 NOT L62
 L134 15 SEA ABB=ON L132 NOT L62
 L135 34 SEA ABB=ON (L131 OR L132)
 FILE 'STNGUIDE' ENTERED AT 16:10:18 ON 16 DEC 2005
 FILE 'MEDLINE, CAPLUS, EMBASE, PASCAL, ESBIODBASE, NIOSHTIC, INSPEC,
 BIOSIS, LIFESCI, TOXCENTER, SCISEARCH' ENTERED AT 16:10:42 ON 16 DEC 2005
 L136 47 DUP REM L130 L129 L128 L135 (20 DUPLICATES REMOVED)
 ANSWERS '1-9' FROM FILE MEDLINE
 ANSWERS '10-26' FROM FILE CAPLUS
 ANSWERS '27-31' FROM FILE EMBASE
 ANSWERS '32-33' FROM FILE PASCAL
 ANSWERS '34-36' FROM FILE ESBIODBASE
 ANSWER '37' FROM FILE NIOSHTIC
 ANSWER '38' FROM FILE INSPEC
 ANSWERS '39-43' FROM FILE BIOSIS
 ANSWERS '44-47' FROM FILE TOXCENTER
 D IALL 1-9
 D IBIB ED ABS HITIND 10-26
 D IALL 27-47
 FILE 'HOME' ENTERED AT 16:11:16 ON 16 DEC 2005

=>

Searched by Barb O'Bryen, STIC 2-2518

L113 E BIOSENS/CT
 E E5+ALL
 7849 SEA ABB=ON BIOSENSOR+NT/CT
 E BIOASSAY/CT
 E E3+ALL
 L114 15116 SEA ABB=ON BIOASSAY/CT
 L115 0 SEA ABB=ON L102 AND L103
 L116 0 SEA ABB=ON (L102 OR L103) AND (L104 OR L105 OR L106 OR L107)
 AND (L108 OR L109 OR L110 OR L111 OR L112 OR L113 OR L114)
 L117 12 SEA ABB=ON (L104 OR L105 OR L106 OR L107) AND L108 AND L109
 L118 1 SEA ABB=ON (L104 OR L105 OR L106 OR L107) AND L108 AND (L113
 OR L114)
 D TRIAL L117 1-12
 L119 578914 SEA ABB=ON IN VITRO STUDY/CT
 L120 0 SEA ABB=ON CELL MONOLAYER/CT
 L121 4485 SEA ABB=ON MONOLAYER CULTURE/CT
 L122 1551 SEA ABB=ON CILIARY MOTILITY/CT
 L123 4 SEA ABB=ON (L104 OR L105 OR L106 OR L107) AND L108 AND L109
 AND (L119 OR (L121 OR L122))
 L124 8 SEA ABB=ON L117 NOT (L118 OR L123)
 D TRIAL 1-18
 L125 38308 SEA ABB=ON QUANTITATIVE ANALYSIS/CT
 L126 1 SEA ABB=ON L117 AND L125

FILE 'STNGUIDE' ENTERED AT 16:04:24 ON 16 DEC 2005

FILE 'CAPLUS' ENTERED AT 16:04:57 ON 16 DEC 2005
 D QUE L5
 D QUE L3

FILE 'MEDLINE' ENTERED AT 16:04:57 ON 16 DEC 2005
 D QUE L95

FILE 'EMBASE' ENTERED AT 16:04:57 ON 16 DEC 2005
 D QUE L115
 D QUE L116.

FILE 'JICST-EPLUS, PASCAL, CABA, BIOTECHNO, ESBIODASE, NTIS, NIOSHTIC,
 ENVIROENG, HEALSAFE, INSPEC, BIOSIS, CONFSCI, LIFESCI, POLLUAB,
 TOXCENTER, CEABA-VTB, WPIX, SCISEARCH' ENTERED AT 16:05:30 ON 16 DEC 2005
 D QUE L62
 D QUE L63

FILE 'MEDLINE, CAPLUS, BIOSIS, SCISEARCH' ENTERED AT 16:05:42 ON 16 DEC
 2005

L127 5 DUP REM L95 L5 L62 (0 DUPLICATES REMOVED)
 ANSWER '1' FROM FILE MEDLINE
 ANSWERS '2-3' FROM FILE CAPLUS
 ANSWER '4' FROM FILE BIOSIS
 ANSWER '5' FROM FILE SCISEARCH
 D IALL 1
 D IBIB ED ABS HITIND 2-3
 D IALL 4-5

FILE 'STNGUIDE' ENTERED AT 16:06:10 ON 16 DEC 2005

FILE 'JICST-EPLUS, PASCAL, CABA, BIOTECHNO, ESBIODASE, NTIS, NIOSHTIC,
 ENVIROENG, HEALSAFE, INSPEC, BIOSIS, CONFSCI, LIFESCI, POLLUAB,
 TOXCENTER, CEABA-VTB, WPIX, SCISEARCH' ENTERED AT 16:08:29 ON 16 DEC 2005
 D QUE L66